Dissertation on

A COMPARATIVE STUDY ON THE CLINICAL PROFILE AND OUTCOME OF ST-ELEVATION MYOCARDIAL INFARCTION AMONG DIABETIC AND NONDIABETIC SOUTH INDIAN PATIENTS

Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI

In partial fulfillment of the regulations

For the Award of the Degree of

M.D GENERAL MEDICINE

(BRANCH-I)

KILPAUK MEDICAL COLLEGE
CHENNAI

MARCH 2007

CERTIFICATE

This is to certify that **"A COMPARATIVE STUDY ON THE CLINICAL PROFILE AND OUTCOME OF ST-ELEVATION MYOCARDIAL INFARCTION AMONG DIABETIC AND NONDIABETIC SOUTH INDIAN PATIENTS"** is bonafide work done by **Dr. SURESH DAVIS**, post graduate student, Department of General Medicine, Kilpauk Medical College, Chennai 10 under my guidance and supervision in fulfillment of regulations of The Tamilnadu Dr. M.G.R. Medical university for the award of M.D. Degree Branch I, Part II (General medicine) during the academic period from March 2004 to March 2007.

Prof. S.R. SAKUNTHALA, M.D.,
Professor and Head
Department of General Medicine
Kilpauk Medical College
Chennai 10.

Dr. A THIAGAVALLI KIRUBAKARAN, M.D.
The Dean
Kilpauk Medical College
Chennai 600 010.

ACKNOWLEDGEMENT

First and foremost I would like to thank **Dr. Thiagavalli Kirubakaran,** Dean, Kilpauk Medical College for permitting me to use the resources and clinical material of this hospital .

I thank **Dr. S.R. Shakunthala**, Professor and Head of department of internal medicine for granting me the permission to conduct this study. I also thank **Dr. K. S. Sai Kumar** retired Professor of department of internal medicine, and assistants **Dr. Rajeshekar, Dr. Jayakumar, Dr. Venkateshvarlu** for their priceless support and guidance .

I also thank **Dr. S. Ramasamy, Dr. A. Joseph Navaseelan and Dr. Kulothungan** for the support extended to me.

I am grateful to **Dr. Dhanapal** Professor Cardiology for his valuable advice and help given to me for this research study.

I am grateful to **Mr. Padmanabhan** Statistician and researcher Indian Council for Medical Research, my fellow post graduates and house surgeons for their invaluable support.

Last but not the least I am grateful to the electrocardiography technician, and the numerous support and sufferings by patients in making this research work a success.

CONTENTS

INTRODUCTION

Heart disease was thought to be associated with diabetes as early as 1883, when Vergeley recommended testing the urine of patients with angina for glucose[1]. However, as more patients with diabetes survived following the discovery of insulin and improvements in the treatment of renal failure and infection, there was a marked increase in morbidity and mortality from cardiovascular disease.

Diabetes mellitus is a strong risk factor for cardiovascular disorders, including coronary heart disease[2,3]. In previous studies, diabetes mellitus has been diagnosed in 10 to 24% of patients with acute myocardial infarction[4,5,6]. Furthermore, the age-adjusted prevalence of diabetes among patients with acute myocardial infarction has increased significantly over the past two decades. A true increase in diabetes, improved documentation in medical records and longer survival of diabetic patients are all factors underlying this increase [7].

Patients with diabetes and myocardial infarction were older[3,8, 9 ,10, 11 ,12,13] and more likely to be females[14]. They present more with anterior wall myocardial infarction, to receive thrombolysis later and to have triple-vessel coronary artery disease[14]. They have more severe coronary artery disease[11] and poor left ventricular ejection fraction[11, 12]. Diabetes mellitus is an independent predictor

for mortality after myocardial infarction[3,10,11,15] and a two to fourfold increase in mortality due to coronary artery disease was noted among diabetics[16]. American Heart association AHA has recently stated that "diabetes is a cardiovascular disease"[17].

Most studies[3,6,10,17] concur that atrioventricular block is more frequent among diabetic than nondiabetic patients, the occurrence of ventricular tachycardia or fibrillation has been less consistent. Some reports[16, 18] suggest that this arrhythmia is more common among patients with diabetes. However, in a recent observational study[19], ventricular fibrillation occurred substantially less frequently among diabetic patients treated with glibenclamide compared with diet or other oral hypoglycemic agents.

AIMS AND OBJECTIVES

1. To study the influence of diabetes on the age of occurrence of STEMI.

2. To study the gender distribution among Diabetic and Nondiabetic patients with STEMI.

3. To study the incidence of painless STEMI in Diabetic and Nondiabetic patients.

4. To study the influence of diabetes on the principal region of myocardial involvement in STEMI.

5. To study the influence of diabetes on complications of STEMI.

6. To evaluate the influence of diabetes on in-hospital mortality in STEMI patients.

REVIEW OF LITERATURE

ST-ELEVATION MYOCARDIAL INFARCTION

DEFINITION

Epidemiological reports from the World Health Organization and American Heart Association beginning in the late 1950s required the presence of at least two of the following : characteristic symptoms, electrocardiographic changes, and a typical rise and fall in biochemical markers for the diagnosis of myocardial infarction[20]. This epidemiological approach was then generally adopted in routine clinical practice, although the rigor with which clinicians apply the electrocardiographic and biochemical criteria for infarction varies considerably.

Advances in the techniques for diagnosing MI, especially the introduction of assays for cardiac-specific troponins, were the impetus for a consensus document published jointly by the European Society of Cardiology and the American College of Cardiology[21].

Revised Definition of Myocardial Infarction MI

Criteria for acute, evolving, or recent MI

Either one of the following criteria satisfies the diagnosis for an acute, evolving, or recent MI :

1. Typical rise and gradual fall troponin or more rapid rise and fall CK-MB of biochemical markers of myocardial necrosis with at least one of the following :
    a. Ischemic symptoms
    b. Development of pathologic Q waves on the ECG reading
    c. ECG changes indicative of ischemia ST-segment elevation or depression
    d. Coronary artery intervention e.g., coronary angioplasty

2. Pathological findings of an acute MI

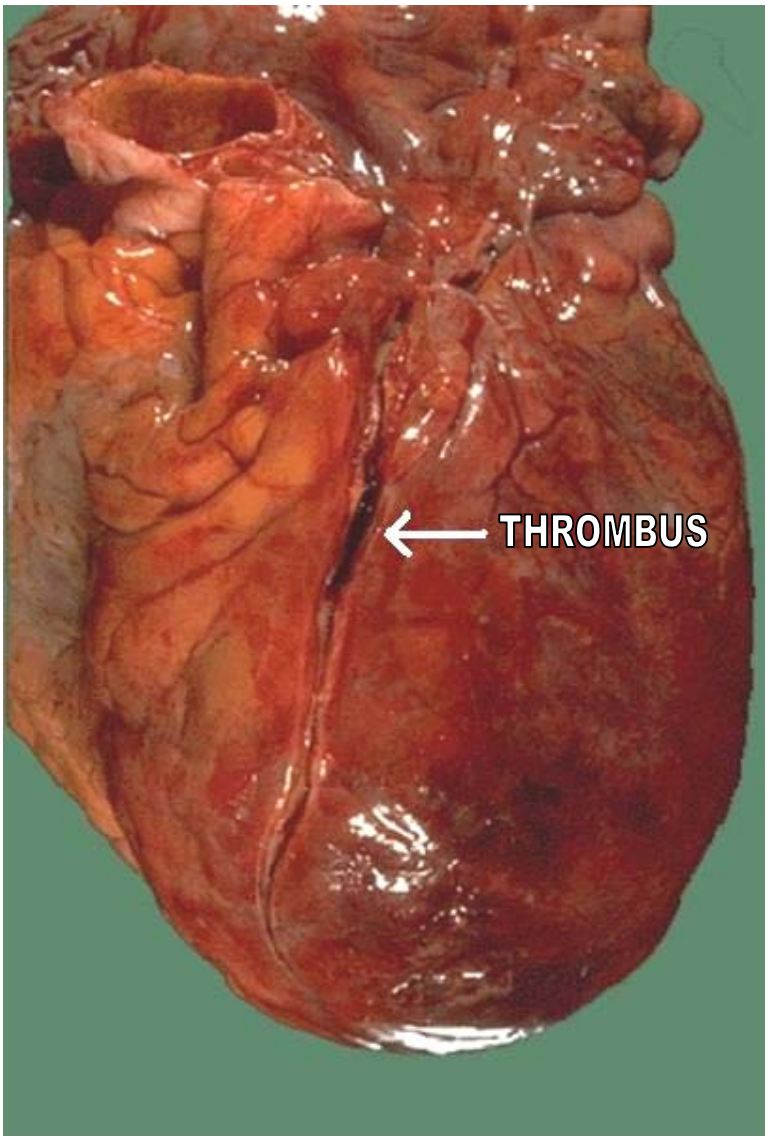
Criteria for established MI

Either of the following criteria satisfies the diagnosis for established MI :

1. Development of new pathological Q waves on serial ECG readings. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized, depending on the length of time that has passed since the infarct developed.

2. Pathological findings of a healed or healing MI

The revised definition of MI has important implications not only for clinical care of patients but also for tracking epidemiological trends, public policy, and clinical trials [22,23].



Coronary thrombus in the Anterior descending coronary artery

Composition of Plaques

The atherosclerotic Plaque is composed primarily of fibrous tissue of varying density and cellularity with superimposed thrombus. Calcium, lipid-laden foam cells, and extracellular lipid each constitutes 5 to 10 percent of the remaining area. The atherosclerotic plaques that are associated with thrombosis and a

total occlusion, located in infarct-related vessels, are generally more complex and irregular than those in vessels not associated with STEMI. Histological studies of these lesions often reveal plaque rupture or erosion. Coronary arterial thrombi responsible for STEMI are approximately 1 cm in length in most cases, adhere to the luminal surface of an artery, and are composed of platelets, fibrin, erythrocytes, and leukocytes. The composition of the thrombus may vary at different levels: a white thrombus is composed of platelets, fibrin, or both, and a red thrombus is composed of erythrocytes, fibrin, platelets, and leukocytes[24]. Early thrombi are usually small and non-occlusive and are composed predominantly of platelets.

Plaque fissuring and disruption

In atherosclerotic plaques prone to disruption, there is an increased rate of formation of metalloproteinase enzymes such as collegenase, gelatinase, and stromelysin that degrade components of the protective interstitial matrix[25]. These proteinases can be elaborated by activated macrophages and mast cells that have been shown to accumulate in high concentration at the site of atheromatous erosion and plaque disruption in patients who died of STEMI[25]. Examination of specimens from atherectomy reveals a much higher content of macrophages and tissue factor in patients with unstable angina or STEMI compared with patients with chronic stable angina[26]. In addition to these structural aspects of

vulnerable or high-risk plaques, stresses induced by intraluminal pressure, coronary vasomotor tone, tachycardia cyclic stretching and compression, and disruption of nutrient vessels combine to produce plaque disruption at the margin of the fibrous cap near an adjacent plaque –free segment of the coronary artery wall shoulder region of plaque[27]. A number of key physiological parameters such as systolic blood pressure, heart rate, blood viscosity, endogenous tissue plasminogen activator t-PA activity, plasminogen activator inhibitor-1 PAI-1 levels, plasma cortisol levels, and plasma epinephrine levels that exhibit circadian and seasonal variations are increased at times of stress. They act in concert to produce a heightened propensity to plaque disruption and coronary thrombosis, yielding the clustering of STEMI in the early morning hours, and especially in the winter and after natural disasters[28].

DIABETES AND CARDIOVASCULAR MORTALITY

Rancho Bernardo Study[29], which followed subjects aged 40 to 79 for 14 years found that death rates were increased in subjects with diabetes, the risk factor-adjusted relative odds were 3.3 in women and 1.9 in men. Factors associated with an increase in mortality rates among those with diabetes include male gender, black race, longer duration of diabetes, and insulin use[30]. Coronary artery and cerebrovascular disease, accounts for 65% of all deaths among persons with diabetes. Patients with type 2 diabetes and

patients with type 1 diabetes have similar causes of death, including CAD and renal failure [31] [32].

Life expectancy is shortened, with diabetic males living, on average, 9.1 years less and diabetic females living 6.7 years less than their nondiabetic counterparts[33]. Haffner and colleagues examined the mortality among 1,000 persons with type 2 diabetes and 1,300 subjects without diabetes and found that the mortality of those with diabetes was similar to that for those without diabetes who had a myocardial infarction MI[34]. These data suggest that caregivers should treat individuals with type 2 diabetes as if they had experienced an MI. Mukamal et al.[35]. Studied 1,935 patients hospitalized with an acute MI and found that the mortality among those with diabetes in the short-term period was similar to that of the patients without diabetes who had an MI previously and twice that of patients without diabetes who had suffered their first acute coronary event. Malmberg et al.[36] evaluated the findings of the OASIS Organization to Assess Strategies for Ischemic Syndromes registry and found that patients with diabetes hospitalized for unstable angina or non-Q-wave MI had the same long-term morbidity and mortality as patients without diabetes with established cardiovascular disease.

Over the past three decades, there have been significant decreases in cardiovascular mortality in the United States. However, the effect on mortality in patients with diabetes has

lagged well behind that in the general population[37]. The death rate among nondiabetic men with CAD decreased by 36.4% as compared to a decrease of 13.1% for diabetic men, and the death rate among nondiabetic women decreased by 27% as compared to an increase of 23% among diabetic women [37].

PREVALENCE AND RISK FACTORS FOR CORONARY ARTERY DISEASE IN TYPE 1 DIABETES

Long-term follow-up of patients with type 1 diabetes has demonstrated that the first cases of clinically manifest CAD occur late in the third decade or in the fourth decade of life regardless of whether diabetes developed early in childhood or during late adolescence. CAD risk increases rapidly after the age of 40, and by the age of 55 years, 35% of men and women with type 1 diabetes die of CAD[32] compared with 8% of those without diabetes. Women with type 1 diabetes lose most of the inherent protection from CAD observed in women without diabetes[32,38,39]. The occurrence of severe coronary artherosclerosis before the age of 55 in a subset of patients with type 1 diabetes regardless of whether diabetes developed in childhood or adolescence suggests that diabetes mainly accelerates the progression of early atherosclerotic lesions that commonly occur, even in the absence of diabetes, at a young age in the general population [32].

Patients with type 1 diabetes followed from the onset of microalbuminuria developed CAD eight times more frequently than patients without microalbuminuria[40]. Krolewski et al [31] reported that the risk of development of CAD in patients with persistent proteinuria was 15 times higher than the risk among those without proteinuria. Microalbuminuria in type 1 diabetes is therefore not only a marker for renal disuse but also a potent marker of CAD risk.

Prevalence and Risk Factors for Coronary Artery Disease in Type 2 Diabetes

Type 2 diabetes increases relative risk of cardiovascular disease two to fourfold compared with the risk in the general population[41,42,43,44]. The increase in cardiovascular risk is particularly high in women. The protection against atherosclerosis in premenopausal women is almost completely lost in women with diabetes[45,46].

Traditional risk factors play an important role in the development of atherosclerosis in subjects with diabetes, the rate of cardiovascular mortality and morbidity in persons with diabetes exceeds by 50% the rate predicted by these risk factors.

Many of these patients with type 2 diabetes have several risk factors for CAD. The term metabolic syndrome was first used by Gerald Reaven in 1988[47] to describe this clustering of risk factors

including hypertension, dyslipidemia, hyperglycemia, and insulin resistance. The National Cholesterol Education Program Adult Treatment Panel III (ATPIII) guidelines for cholesterol management in 2001 recognized that the metabolic syndrome is a collection of the risk factors mentioned above, as well as abdominal obesity [48].

PATHOPHYSIOLOGY OF DIABETIC CARDIOVASCULAR COMPLICATIONS

Insulin levels, Insulin resistance and Hyperglycemia

Insulin resistance that is present many years or more before the clinical onset of overt diabetes is associated with other atherogenic risk factors, such as hypertension, lipid abnormalities, and a procoagulant state[49,50,51,52,53,54,55]. Several studies have shown an inverse correlation between insulin sensitivity and atherosclerosis[56,57,58]. The Bruneck Study database suggest[59] that these risk factors are present in 84% of patients with type 2 diabetes. Thus, an increased prevalence of CAD is apparent in patients with impaired glucose tolerance[42,44,60] and in those with newly diagnosed type 2 diabetes[61,62]. The duration of insulin resistance among hyperglycemic and diabetic individuals probably contributes to the development of atherosclerosis. However no obvious association between the extent or severity of macrovascular complications and the duration or severity of type 2 diabetes[38, 63]

has been found, most likely because the duration of insulin resistant is often unknown.

Another possibility is that the serum insulin level and not insulin resistance has direct cardiovascular effects. Despres and colleagues[64] followed 2,000 diabetic men without clinically overt CAD for 5 years and found that those who had a cardiovascular event had serum insulin levels that were 18% higher than those in controls.

Serum glucose levels may be an important risk factor for cardiovascular disease. Andersson and Svardsudd[65] demonstrated that fasting serum glucose levels are independently related to all-cause and cardiovascular mortality. The San Antonio Heart Study[66] showed similar findings for subjects in the highest quartile of fasting glucose levels, who had a 4.7 times greater risk of cardiovascular disease than did those in the first two quartile levels combined.

The direct relationship between glucose levels and cardiovascular disease also is seen in patients with type 1 diabetes. A 1% increase in levels of glycosylated hemoglobin doubled the increase in cardiovascular disease [67]. Several studies have shown a direct relationship with the serum glucose levels on clinical events, including MI and strokes, with glucose levels ranging from an abnormal glucose tolerance test to frank diabetes[68,69,70]. This graded

effect of serum glucose levels on clinical events may be due in part to a direct effect on the vasculature, as evidenced by a similar direct relationship of serum glucose levels to the intima-media thickness of the carotid (as a marker for the presence and degree of atherosclerosis). The Atherosclerosis Risk in Communities (ARIC) study demonstrated that fasting glucose tolerance was directly related to carotid wall thickness in individuals free of symptomatic cardiovascular disease[71].

The level of chronic hyperglycemia, as determined by measurements of glycosylated hemoglobin, may also be an independent risk factor for coronary heart disease, particularly in women[72,73]. Recent prospective studies demonstrated that microalbuminuria in patients with type 2 diabetes is also an independent predictor of increased cardiovascular mortablity[74, 75]. Insulin resistance may play an important role as a risk factor in the development of diabetic cardiovascular disease. Hyper-insulinemia may be the mechanism by which the effect of hyperglycemia results in atherosclerosis. Insulin level is elevated patients with the metabolic syndrome. The possibility that insulin resistance could result in an increase in cardiovascular disease was first demonstrated in population studies that showed an association between fasting insulin levels and cardiovascular mortality [56,76,77,78]. The relationship of insulin levels and cardiovascular disease is further strengthened by basic research studies that

showed the effect of insulin on various possible mediators for the development of atherosclerosis, specifically the increase in PAI-I and the mitogenic effect on smooth muscle cells *in vitro*[79].

Dyslipidemia

An important mechanism for the development of diabetic atherosclerosis is dyslipidemia. The central feature of diabetic dyslipidemia is increased levels of VLDL due both to increased production of VLDL and to decreased catabolism of triglyceride-rich lipoproteins, including chylomicrons. The increase in hepatic production of VLDL occurs in response to increased delivery of fatty acids from (a) decreased free fatty acid uptake from the striated muscle and (b) increased delivery of the free fatty acids from the increased adipose tissue associated with central obesity.

The increase in triglyceride-rich lipoproteins accumulates not only because of increased VLDL production but also because of decreased catabolism of triglyceride lipoproteins. Lipoprotein lipase, which plays an important role in the metabolism of triglyceride-rich lipoproteins and in particular chylomicrons, is decreased in uncontrolled type 2 diabetes.

The increased level of triglyceride-rich lipoproteins provides an increase in substrate for the cholesterol ester transfer protein.

This promotes the flux of cholesterol from HDL particles, which results in decreased HDL levels, a common finding in type 2 diabetes.

One mechanism of the protective effect of HDL against atherosclerosis may be its ability to prevent oxidation of LDL. There may be qualitative differences in HDL from patients with poorly controlled diabetes that may make it a less effective antioxidant than HDL from normal individuals[80].

The dyslipidemia of diabetes is not characterized by marked elevations of LDL, there are differences in the LDL type found in patients with type 2 diabetes. Specifically, the LDL is smaller and denser than typical LDL particles[81], have a greater tendency to undergo oxidation, which accelerates the atherosclerotic process.

Increased Oxidative Stress in Diabetes

There is recent evidence that increased oxidative stress in diabetes contributes to the development of diabetic complications. This increased stress may be due in part to the decreased availability of antioxidants such as ascorbic acid, vitamin E, uric acid, and glutathione. In addition, there may be an increase in lipid peroxidation products and superoxide anion products, which may be lead to altered vascular function [82,83,84].

Increase in oxidative stress may be the result of several pathways, including advanced glycation end product (AGE) production; small, dense LDL formation; altered polyol activity; or imbalance in the redox state [85]. The activation of this polyol pathway is due to the conversion of glucose to sorbitol via aldolase reductase, which has been associated with microvascular complications [86,87].

Advanced Glycation End Products in Diabetes

AGEs occur as a result of the nonenzymatic glycation of both lipids and proteins. Initially, a labile covalent bond develops between the aldehyde of the glucose molecule and the amino acid side chain on both sugars and lipids. Specifically, glucose is covalently bound mainly to lysine residues in proteins, forming fructose-lysine residues. This reaction results in the development of a Schiff base, which, in turn, undergoes another chemical reaction to form a ketoamine, termed an Amadori product. These products result in cumulative oxidative damage to proteins. These products include CML[88] and pentosidine[89]. The increased levels of pentosidine and CML correlate with the severity of diabetic complications, including nephropathy, retinopathy, and vascular disease. One such Amadori product is glycated (or glycosylated) hemoglobin A1c (HbA1c), which is commonly used to monitor

glycemic control in diabetic patients. Since both free-radical oxidation and glycation are involved, these substances are also called glyoxidation products.

AGEs cross-link to the proteins composing the extracellular matrix and vascular basement membrane, which results in reduced solubility and decreased enzymatic digestion [90,91]. AGE formation also prevents proper assembly of basement proteins, thereby altering their function. This in turn may alter the ability of cells to bind to their substrates.

Enhanced glycation, oxidation, and glyoxidation of lipoproteins have been postulated as a possible cause for the development of diabetic macrovascular disease. Certainly there are increased levels of AGE-modified LDL-apoprotein and LDL-lipid in persons with diabetes relative to levels in persons without diabetes [92]. This would suggest that even in the face of similar glycemic control and other cardiovascular risk factors, the development of diabetic vascular complications would depend on differences of oxidative stress as well as on the tissue level of antioxidants.

Thrombosis and Fibrinolysis in Diabetes

Plaque disruption with overlying thrombosis is a major cause of acute coronary syndromes, including MI, sudden death. There are three underlying mechanisms for this prothrombosis:

heightened platelet reactivity, increased procoagulant activity, and decreased antithrombotic and fibrinolytic activity.

The platelets of diabetic individuals appear to have an increased adherence to the vessel wall and increased circulating platelet mass[93]. Platelet aggregometry studies that measure in vitro platelet reactivity have demonstrated increased aggregation of platelets in response to the agonists ADP, collagen, and thrombin and even spontaneous aggregation of platelets without any agonist[94,95,96,97,98]. Assessment of platelet reactivity in vivo by measurement of blood or urine metabolites released from activated platelets such as thromboxane B2 has shown increased reactivity relative to that of normal healthy controls [94,95].

Patients with diabetes have increased concentrations of fibrinogen, von Willebrand factor, and factor VII [99,100,101]. The level of serum fibrinogen correlates with the levels of proinsulin and insulin in the blood [102].

Several reports indicate that the activity of antithrombotic factors, including protein C and antithrombins, are decreased in subjects with diabetes, which further potentiates the hypercoagulable state [103,104,105,106].

Fibrinolysis is also impaired in individuals with diabetes, particularly those with type 2 diabetes [107,108]. This impairment may be due to the increased activity of PAI-1 in the blood, which

counteracts the action of native tissue plasminogen activator (t-PA) to induce fibrinolysis. PAI-1 is elevated not only in resting states but also in response to physiologic stimuli. The serum level of PAI-1 may be elevated as a result of several factors, including elevated serum levels of insulin, lipids, and glucose[109].

Endothelial Function and Diabetes

Alterations in endothelial function may play an important role in the development of diabetic complications.

The vascular endothelium has been shown to be important in modulating blood cell-vessel wall interaction, regulating blood flow, angiogenesis, lipoprotein metabolism, and vasomotion. An important mediator in maintaining vascular homeostasis is endothelium-derived relaxing factor (EDRF)[110] which has since been found to be nitric oxide[111]. The release of nitric oxide activates soluble guanylate cyclase, resulting in the formation of cyclic guanosine monophosphate (cGMP), which, in turn, activates cGMP-dependent protein kinases, resulting in relaxation of vascular smooth muscle[112,113,114,115]. Alterations in the expression, release, or activity of EDRF may play an important role in the initiation and progression of both micro-and macrovascular disease. Several studies have shown that endothelial-dependent vasodilator function is impaired in patients with type 1 diabetes without hypertension and dyslipidemia[116]. This impairment is in contrast to

that in patients with type 2 diabetes, who have an impairment of both endothelial – dependent and endothelial-independent (smooth muscle) vasodilator function [117,118].

Acute hyperglycemia impairs endothelial-dependent vasodilatation in both macro-and microvessels[119]. Insulin also may play a role. Insulin results in vasodilatation due in part to nitric oxide production. Glucose-clamp experiments with insulin infusion have shown that subjects with type 2 diabetes have little improvement in endothelial-dependent vasodilatation relative to that in subjects without diabetes[119]. As stated previously, there appears to be an increase in oxygen-derived free radicals in the diabetic state. Several studies have shown that high doses of vitamin C can improve endothelial-dependent vasodilatation in patients with both type 1 and type 2 diabetes[120,121]. Intensive lipid lowering by Statin therapy does not improve vasoreactivity in patients with type 2 diabetes, suggesting that mechanisms other than dyslipidemia are responsible for endothelial dysfunction [122].

Silent Ischemia

The propensity of patients with diabetes to present with either silent or unrecognized MI is well established[123,124]. Atypical symptoms such as confusion, dyspnoea, fatigue, or nausea and vomiting were the presenting complaint in 32% to 42% of patients with diabetes with MI compared with 6% to 15% of patients

without diabetes[123,125]. Several groups have reported that the detection of silent ischemia by various noninvasive techniques, including treadmill exercise testing[126,127], ambulatory holter monitoring[128] and exercise thallium scintigraphy[129,130,131,132], is more common in patients with diabetes than in those without diabetes. This finding, however, is not supported by all studies[133,134].

A plausible explanation for painless infarction and ischemic episodes in patients with diabetes is autonomic neuropathy with involvement of the sensory supply to the heart. In autopsies of patients with diabetes who died of silent MIs, typical diabetic neuropathic changes were found in the intracardiac sympathetic and parasympathetic fibers[135], and several studies correlated abnormalities in autonomic function in patients with silent ischemia[126,128,130,136]. The anginal perceptual threshold-the time from the onset of myocardial ischemia (assessed by ST segment depression) to the onset of chest pain during exercise testing -is prolonged in patients with diabetes compared with those without diabetes. This delay in the perception of pain may be related to the impairment of autonomic nervous function[136].

Acute Coronary Syndromes in Patients with Diabetes

Acute ischemic events represent a major cause of death in the diabetic population[61]. Diabetic patients who suffer an MI have a

higher mortality than nondiabetic patients both in the acute phase and on long-term follow-up. Numerous studies have shown that in-hospital mortality rates from MI in patients with diabetes are 1.5 to 2-fold higher than in patients without diabetes[137,138,139,140]. Diabetes remains an independent predictor for a poor prognosis in the thrombolytic era. In the Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) trials, the in-hospital mortality rate was nearly twice as high in patients with diabetes, with more congestive heart failure and twice the rate of clinically recognized reinfarction [137]. In the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial, mortality at 30 days was highest among patients with diabetes treated with insulin (12.5%) compared with patients with diabetes not treated with insulin (9.7%) and nondiabetic (6.2%) patients ($p<0.001$)[141]. Similar results have been reported from the other large studies[142,143,144]. Diabetes is also a risk factor for cardiogenic shock in the setting of acute ischemic syndromes[145]. Overall, despite the overall improvement in survival from an acute MI with thrombolysis, the in-hospital mortality rates in patients with diabetes remain 1.5 to 2 times higher than in patients without diabetes [141,144].

This increased in-hospital mortality among patients with diabetes with acute MI is due predominantly to an increase in the incidence of congestive heart failure [138,140,146,147] although increases

in the incidence of reinfarction, infarct extension, and recurrent ischemia have also been reported [138,139,140,147,148].

Studies using serial determinations of total creatine kinase activity[146,147] radionuclide ventriculography[149], or echocardiography have found no evidence that patients with diabetes sustain more extensive infarctions than their nondiabetic counterparts[150]. Thus, congestive heart failure and cardiogenic shock are more common and more severe in subjects with diabetes than would be expected from the size of the index infarction[144,146,147,149,151,152]. The observation that clinical manifestations of heart failure occur in patients with diabetes despite a modest decrease in left ventricular ejection fraction (EF) led to the suggestion that preexisting diastolic dysfunction is a major culprit in the congestive symptoms[140]. Indeed, subclinical diabetic cardiomyopathy, which is characterized by diastolic dysfunction[153], is likely to be an important factor in this setting.

It should be emphasized, however, the reductions in both left ventricular EF[149,154] and the regional EF of the noninfarcted myocardium[137,149,153] have been well documented in patients with diabetes following MI as compared with patients without diabetes. For example, early angiography in the TAMI trials has demonstrated worse ventricular function in the noninfarcted zone in patients with diabetes [137].

The performance of the left ventricle following MI is determined largely by the extent of coronary disease[155] and the quality of collateral circulation. Thus, the diffuse nature of coronary atherosclerosis in diabetes may contribute to systolic dysfunction of the noninfarcted myocardium. Moreover, a recent study has shown that patients with diabetes have a reduced ability to develop collateral blood vessels in the presence of CAD[156], a finding that also may explain the more frequent occurrence of postinfarction angina and infarct extension[139,140,148,150].

Patients with diabetes surviving MI also suffer higher late mortality rates than patients without diabetes[140,148,157,158,159]. Late mortality is related primarily to both recurrent MI and the development of new congestive heart failure [144,150,158,159,160].

CONGESTIVE HEART FAILURE AND DIABETES

CAD is the most common cause of congestive heart failure in the overall U.S.[161], and diabetic [162] populations. Diffuse CAD can lead to nontransmural infarction with patchy necrosis and myocardial fibrosis, resulting in an impairment of systolic function. Myocardial ischemia may result not only in systolic dysfunction but also in diastolic dysfunction[163,164]. In the setting of an acute MI, patients with diabetes have been reported in some studies to develop heart failure up to 50% of the time[165]. The GUSTO-1 trial demonstrated that heart failure developed in 27% of the subjects

with type 1 diabetes compared with 20% of those with type 2 diabetes and 15% of those in the nondiabetic group. This amounts to the occurrence of heart failure almost twice as frequently in the diabetic population relative to the nondiabetic population[140]. Persons with diabetes are also almost twice as likely as those without diabetes to develop heart failure as a result of an acute coronary syndrome (7.2% vs. 3.8%).

The presence of heart failure in the diabetic population is associated with a poorer long-term prognosis. This GUSTO-1 study demonstrated that cardiac mortality at 30 days in the subjects with type 1 diabetes was 12.5% compared with 9.7% in those with type 2 diabetes and 6.2% in those without diabetes. This poorer outcome was not the result of a larger MI but may have been due to response of the noninfarcted myocardium to the infarct. The MILIS database demonstrated that the prognosis of patients with diabetes was worse relative to that of patients without diabetes (4-year cardiac mortality rates of 25.9% in those with diabetes and 14.5% in those without diabetes), despite the presence of smaller infarcts [as measured by peak creatine phosphokinase (CPK) or area under the curve] and fewer Q-wave infarcts[140]. The GUSTO-1 study also found that vessel patency after MI did not explain this worse prognosis either, since there were similar degrees of infarct-related patency at 90 minutes in the patients with or without diabetes[166].Compensatory hyperkinesis of noninfarcted walls,

which is frequently found in subject without diabetes immediately after MI, is often blunted in the patient with diabetes[166]. This may account for the increased incidence of heart failure.

Materials and Methods

MATERIALS AND METHODS

Setting

A randomised comparative study and analysis with patients drawn from Intensive coronary care unit.

Study Population

The study was conducted over a 2 year period from June 2004 to June 2006. Cases were drawn from Intensive coronary care unit, Department of Cardiology, Kilpauk Medical College. Informed consent was taken from all participants of the study. The clinical profiles of patients admitted with ST elevation MI (STEMI) were analyzed. The patients were grouped into diabetics and nondiabetics based on their previous history of diabetes. A total of 2113 patients were initially chosen for the study. 671 were found to be diabetic and the remaining 1442 were nondiabetic. On application of exclusion criteria, 458 and 1036 patients were excluded from the diabetic and nondiabetic group respectively. Eight of the 113 diabetic patients and 14 among the 406 nondiabetic patients expired as a result of cardiogenic shock soon after admission. 9 and 6 patients expired due to other complications of acute myocardial infarction in the diabetic and nondiabetics groups respectively. In the above patients blood sugar values and echo evaluation could not be performed.

Inclusion Criteria

Patients with STEMI evidenced by ECG and clinical symptoms <24 hrs

Patients with age above 20 years were chosen for the study.

Exclusion Criteria

- Patients with previous history of hypertension.
- Patients with previous history of MI.

Data Collection and Assessment

Information collected consisted of basic data, including name, sex, age and occupation. The nature of habitat, whether sedentary lifestyle or manual labourer, was recorded. Main presenting complaint at the time of admission was recorded from the patients.

Previous history of diabetes was sought including the duration and regularity of treatment. Patients meeting the exclusion criteria were excluded from the study by detailed history and clinical examination of all systems.

Vitals were recorded.

Cardiovascular examination of patients done and patients classified according to Killips classification.

Random blood sugar and ECG was taken at the time of admission. The region of myocardial damage was identified with the help of ECG.

Patient was then treated for the acute coronary event (viz. thrombolysed or anticoagulated) Fasting blood sugar was sent the following morning.

All complications including mortality following the myocardial infarction during the stay in ICCU were noted.

Echocardiography was performed at the time of discharge and ejection fraction calculated.

Statistical Methods

The data obtained from the study was analyzed for statistical significance using the Students t test, Chi square and Fischer's Test. Values were expressed as mean and standard deviation. Calculation was done using SPSS software with the assistance of our college statistician and ICMR research worker

Reference Criteria Used In This Study

ST Elevation Myocardial Infarction

The ECG criteria for the diagnosis of STEMI as outlined in the MILIS study are the presence of any one of the following in the setting of chest pain: (1) new or presumably new Q waves (at least

30 ms wide and 0.20 mv deep) in atleast two leads from any of the following (a) leads II, III or AVF ;(b) leads V1 through V6 or (c) leads I and AVL ;(2) new or presumably new ST-T segment elevation(>0.10 mv measured 0.02 s after the J point in two contiguous leads) or (3) a complete left bundle branch block in the appropriate clinical setting [167].

Localization of Region of Myocardial Infarction in ECG

- Extensive Anterior wall (AWMI) – Reflected by typical infarction pattern in standard lead I, AVL and all precordial leads

- Anteroseptal wall (ASMI) – Reflected by infarction pattern in leads $V_1 – V_4$.

- Anterolateral wall (ALMI) – Reflected by infarction pattern in leads I, AVL &$V_4 – V_6$.

- Inferior wall (IWMI) – Reflected by infarction pattern in leads II,III & AVF[168].

Killip Classification

Based on clinical examination[169]

Class	Definition
I	No signs of pulmonary or venous congestion
II	Moderate heart failure evidenced by rales at lung base, S3, Tachypnea or Signs of Rt. heart failure
III	Severe heart failure, Pulmonary edema
IV	Shock with systolic blood pressure <90mm Hg, Signs peripheral vasoconstriction, Peripheral cyanosis, Mental confusion. Oliguria

Ventricular Tachycardia

A series of 3 or more consecutive ventricular ectopic beats that are recorded in rapid succession[170].

Sustained Ventricular Tachycardia

Ventricular tachycardia that persists for >30s or requires termination due to hemodynamic collapse[171].

Ventricular Fibrillation

ECG showing completely irregular, chaotic and deformed deflections of varying height, width and shape[170].

Complete Heart Block

Complete interruption of AV conduction evidenced by AV dissociation and slow ventricular rate[172].

Blood Glucose Estimation

Method : In this study we used GOD/POD method for estimation of fasting and random blood sugar.

Principle : Glucose is oxidized by the enzyme Glucose Oxidase (GOD) to give D-Gluconic acid and hydrogen peroxide. Hydrogen peroxide in presence of enzyme Peroxidase (POD) oxidizes phenol which combines with 4 aminoantipyrine to produce a red colored quinonimine dye. The intensity of the color developed is proportional to glucose concentration in the sample.

$$\text{D- glucose} + H_2O + O_2 \xrightarrow{\text{GOD}} \text{D – gluconic acid} + H_2O_2$$

$$H_2O_2 + 4\ \text{Amonoantipyrine} + \text{Phenol} \longrightarrow \text{Red Quinonimine dye} + H_2$$

Reagents

1. Enzyme reagent
2. Buffer solution
3. Glucose standard 100 mg%

Normal Values

Fasting : 70– 100 mg / dl – Normal

>100 – 125 mg/dl – Impaired fasting glucose

≥ 126 mg/dl – Diabetes mellitus

After 2 hours of injection of 75 gms glucose.

<140 mg/dl- Impaired Glucose tolerance test

>200 mg/dl – diabetes mellitus[173].

Observation and Analysis

OBSERVATION AND ANALYSIS

PATIENTS FROM CARDIOLOGY OP

Diabetic : 113

Non-diabetic : 406

TABLE – 1

DISTRIBUTION ACCORDING TO AGE

Age	Diabetic		Non Diabetic	
	No.	Percentage	No.	Percentage
≤ 30	0	0	7	1.72
31-40	16	14.16	58	14.30
41-50	22	19.47	121	29.80
51-60	36	31.86	115	28.32
61-70	31	27.43	70	17.24
> 70	8	7.08	35	8.62
Total	**113**	**100%**	**406**	**100%**

TABLE – 2

MEAN AND STANDARD DEVIATION

Group	Number	Mean	S.D.	Significance
Diabetic	406	53.23645	± 12.13701	P > 0.05 NS
Non Diabetic	113	55.0531	± 10.98606	

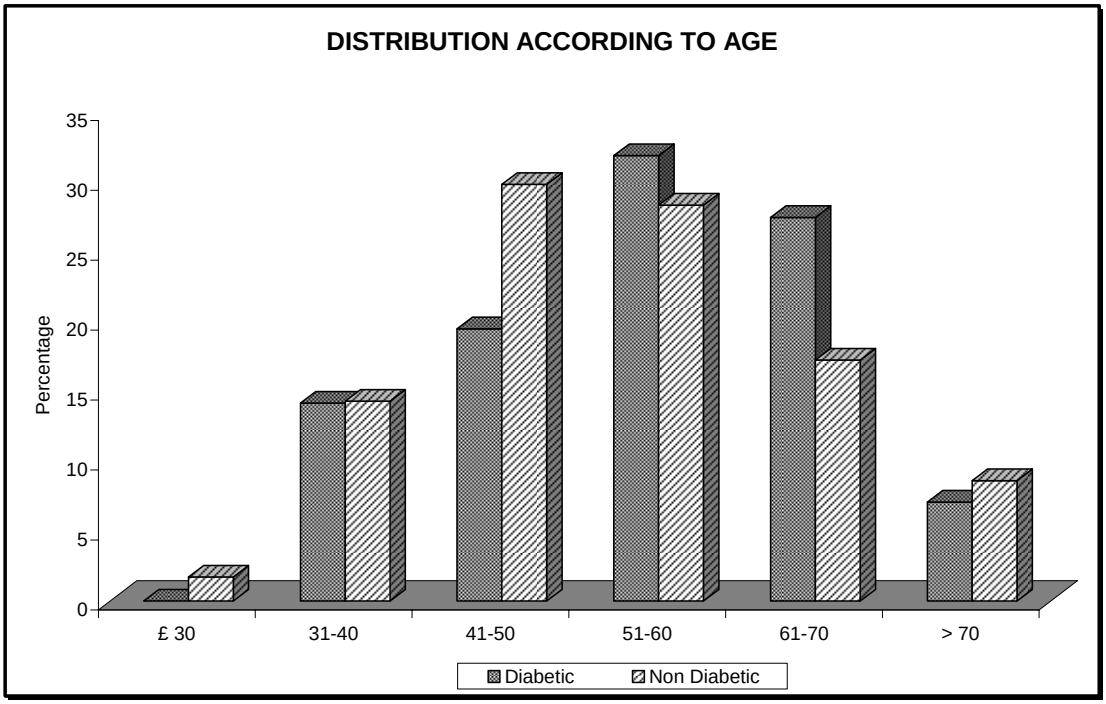

DISTRIBUTION ACCORDING TO AGE
Percentage
35
30
25
20
15
10
5
0
£ 30
31-40
41-50
51-60
61-70
> 70
Diabetic
Non Diabetic

TABLE - 3

DISTRIBUTION ACCORDING TO GENDER

Sex	Diabetic		Non Diabetic	
	No.	Percentage	No.	Percentage
Female	33	29.20	66	16.25
Male	80	70.80	340	83.75
Total	**113**	**100%**	**406**	**100%**

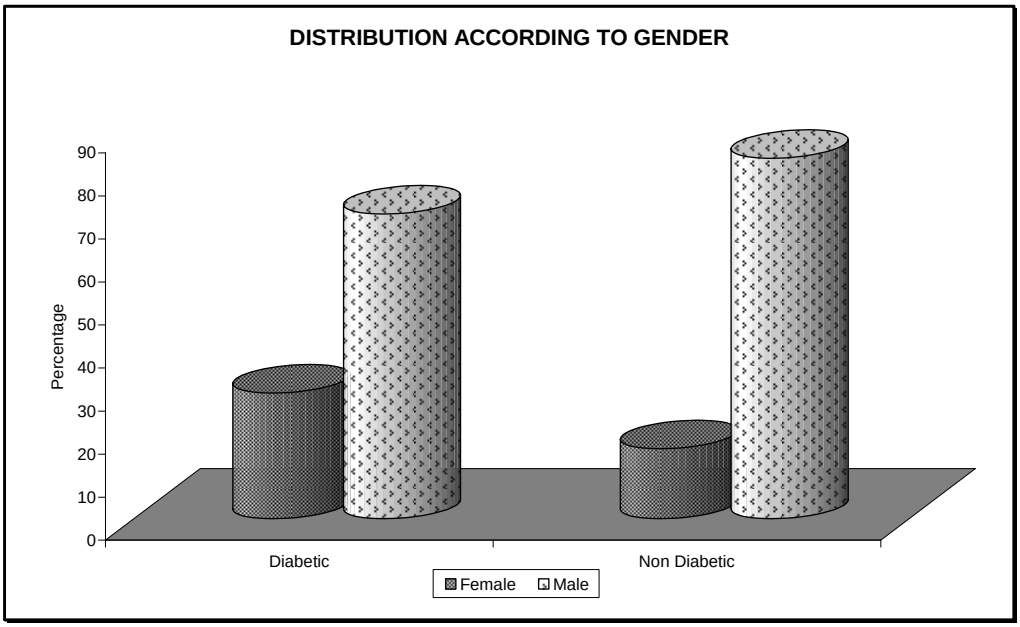


TABLE - 4

DISTRIBUTION ACCORDING TO OCCUPATION

Occupation	Diabetic		Non Diabetic	
	No.	Percentage	No.	Percentage
Manual Labour	65	57.52	295	72.60
Sedentary	48	42.48	111	27.40
Total	**113**	**100%**	**406**	**100%**

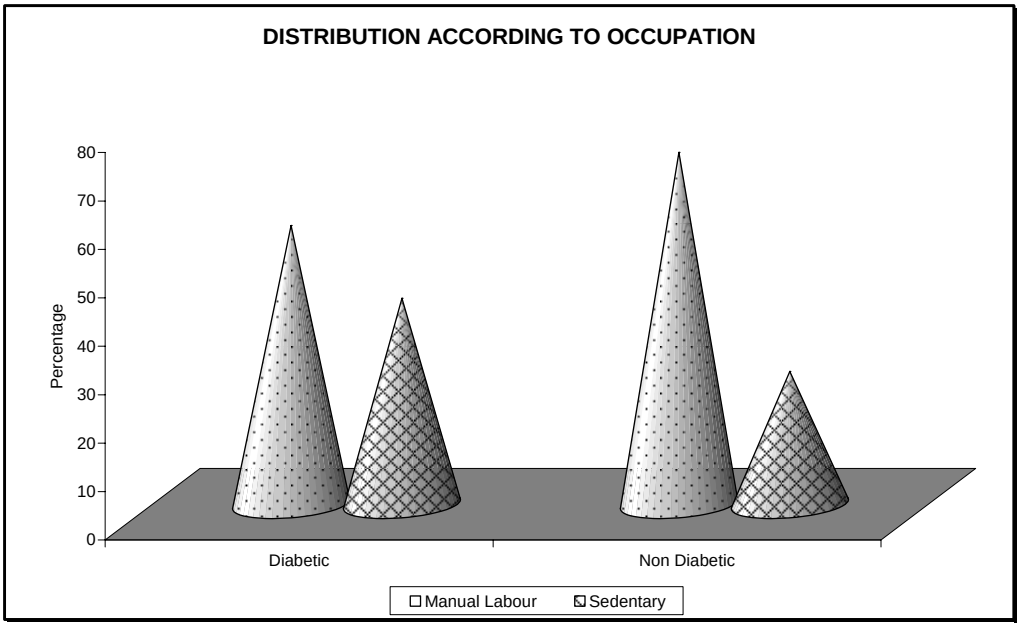


TABLE - 5

DISTRIBUTION ACCORDING TO THE PRESENCE OR ABSENCE OF CHEST PAIN

Group	Chest Pain		Total
	Present	Absent	
Diabetic	90	23	113
Non Diabetic	392	14	406

$\chi^2 = 38.08$

P value < 0.001 (Significant)

Odds Ratio : 7.16

Non chest pain presentation was 7.16 times more common in diabetic compared to Non diabetics.

TABLE - 6

DISTRIBUTION ACCORDING TO THE DURATION OF DIABETICS

Duration	Number	Percentage
< 5 years	70	61.95
5-10	41	36.28
> 10 years	2	1.77
Total	**113**	**100%**

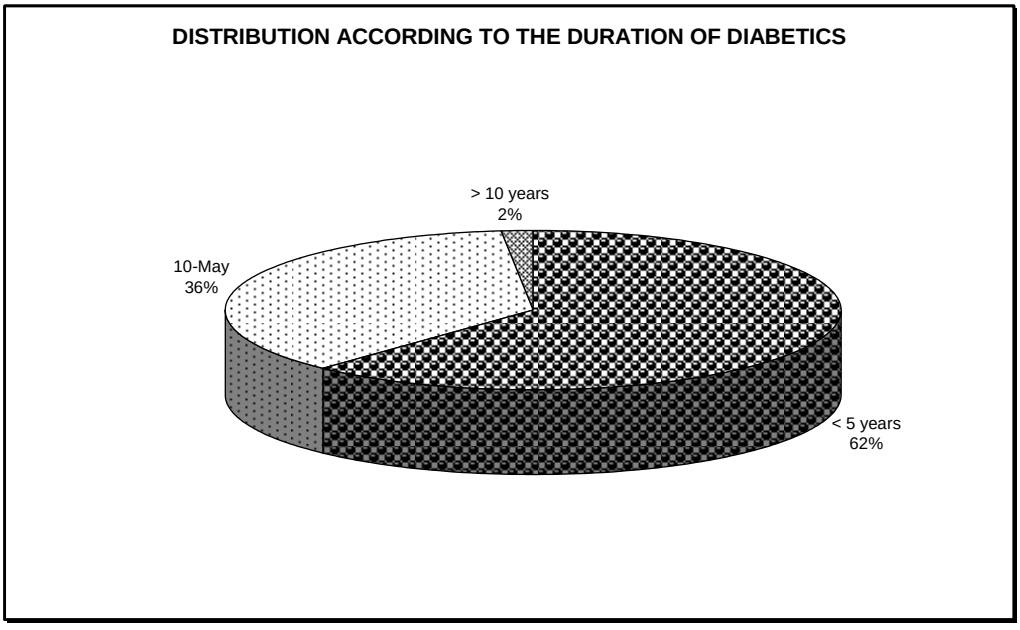


TABLE - 7

DISTRIBUTION ACCORDING TO REGULARITY OF DIABETIC TREATMENT

Treatment	Number	Percentage
Regular	58	51.32
Irregular	55	48.68
Total	**113**	**100%**

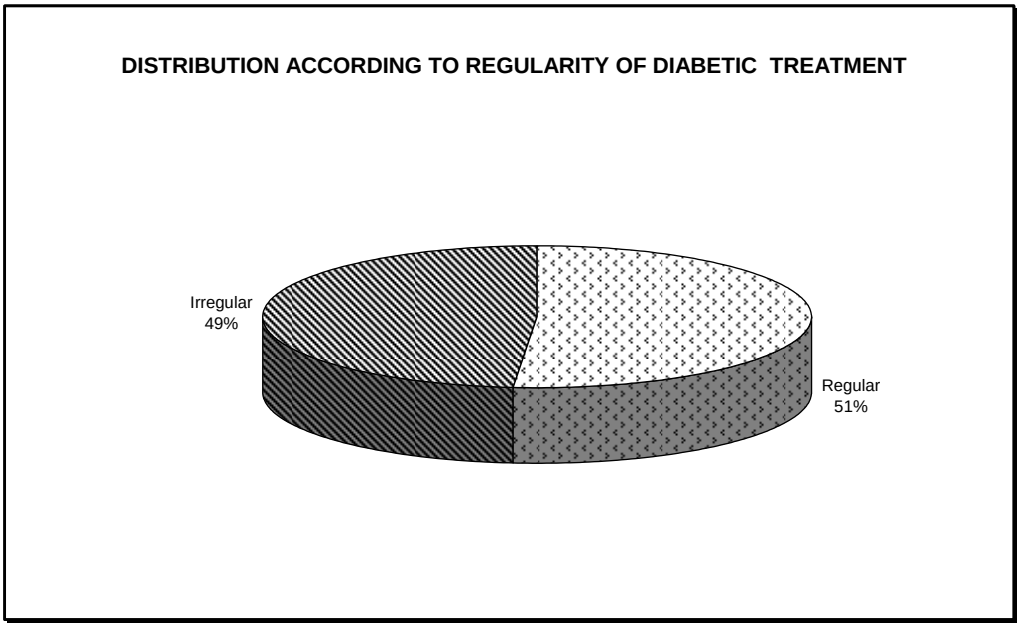


TABLE - 8

MEAN AND STANDARD DEVIATION OF FASTING AND RANDOM BLOOD SUGARS IN DIABETIC PATIENTS

Blood Sugar	Mean	SD
Fasting	175.61	± 54.05
Random	252.50	± 76.40

TABLE - 9

DISTRIBUTION ACCORDING TO KILLIP CLASSIFICATION

Killip	**I**	**II**	**III**	**IV**	**Total**
Diabetic	66	36	3	8	406
Non Diabetic	371	19	2	14	113

$\chi^2 = 80.07$

P value <0.001 significant

Diabetic patients showed higher Killip class when compared to non diabetics.

TABLE - 10

DISTRIBUTION ACCORDING TO THE WALL INVOLVED IN MYOCARDIAL INFARCTION

Wall	Diabetic	Non Diabetic
AWMI	96	243
Others	17	163
Total	**113**	**406**

$\chi^2 = 24.54$

P value < 0.001 significant

Odds ratio : 3.79

Diabetic patients had 3.79 times anterior wall myocardial infarction compared to non diabetic patients.

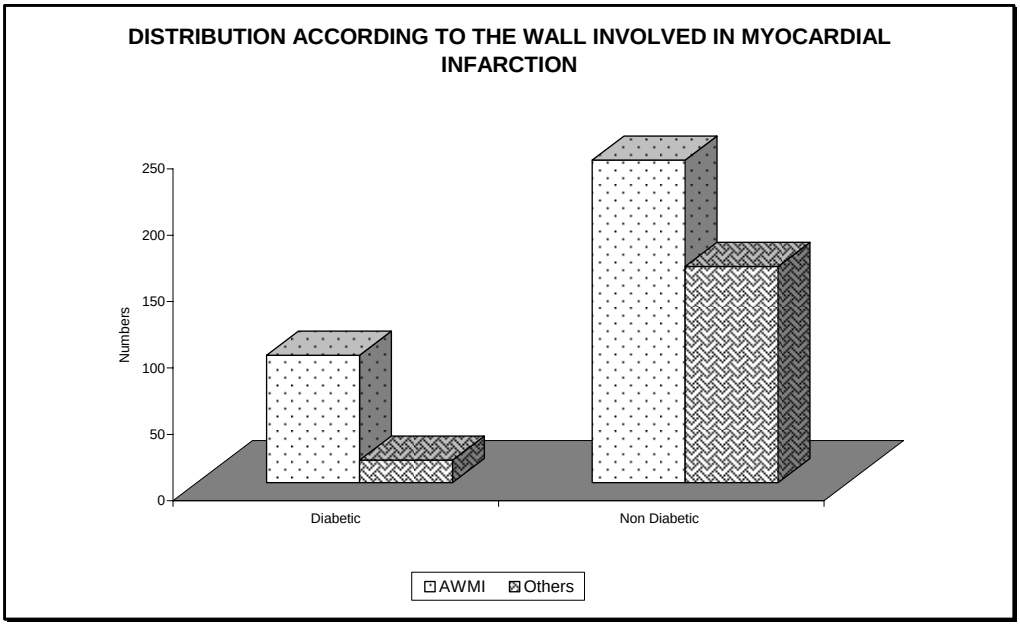


TABLE - 11

DISTRIBUTION ACCORDING TO EJECTION FRACTION

Ejection Fraction	Diabetic		Non Diabetic	
	No.	Percentage	No.	Percentage
≤ 40	9	8.82	2	0.51
41-50	44	43.14	23	5.88
≥ 51	49	48.04	366	93.61
Total	**102**	**100%**	**406**	**100%**

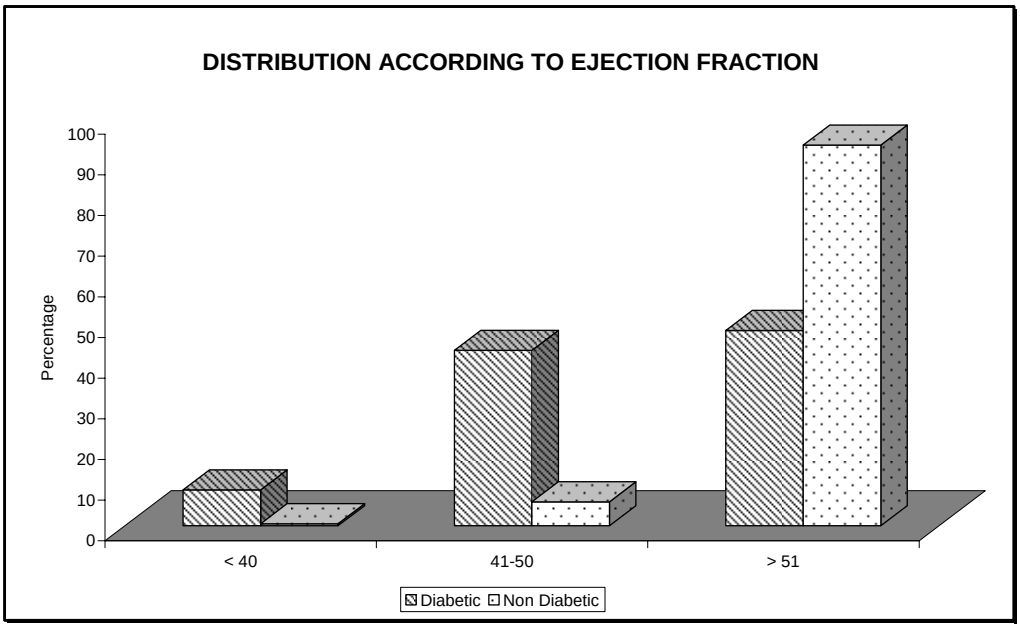


TABLE - 12

DISTRIBUTION ACCORDING TO THE PRESENCE OR ABSENCE OF CARDIAC FAILURE

Group	Congestive Cardiac failure		Total
	Present	Absent	
Diabetic	44	69	113
Non Diabetic	40	366	406

$\chi^2 = 55.02$

P value < 0.001 significant

Odds ratio : 5.83

Cardiac failure was 5.83 times more common in diabetics compared to non diabetics.

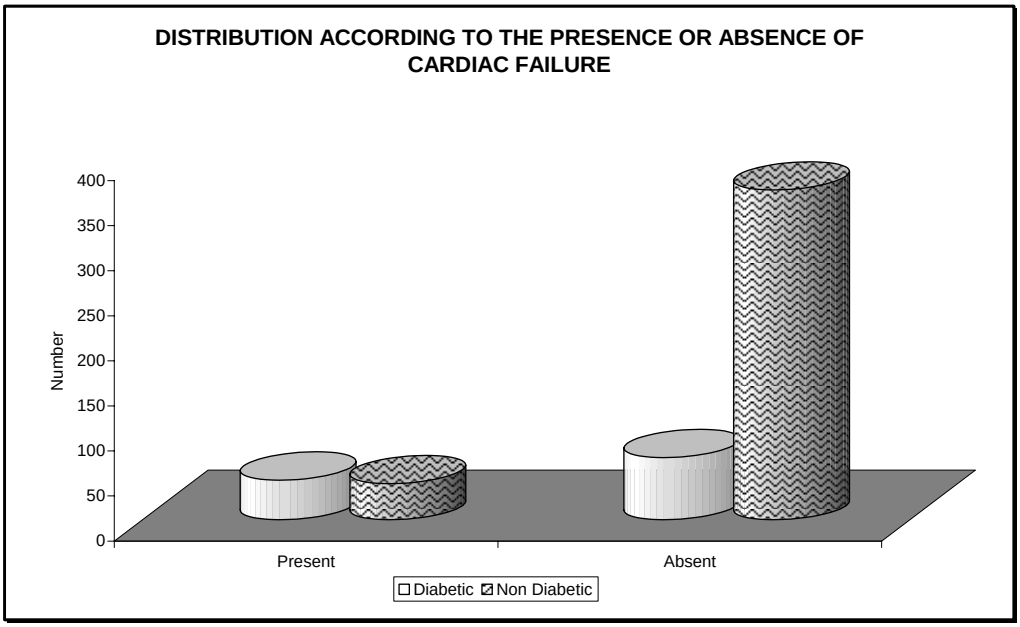


TABLE - 13

DISTRIBUTION ACCORDING TO THE PRESENCE OR ABSENCE OF VENTRICULAR ARRYTHMIAS

Group	Ventricular arrhythmias		Total
	Present	Absent	
Diabetic	7	106	113
Non Diabetic	6	400	406

$\chi^2 = 8.04$

P value = 0.0045 significant

Odds ratio : 4.40

Ventricular arrhythmias were 4.4 times more common in diabetics compared to non diabetics.

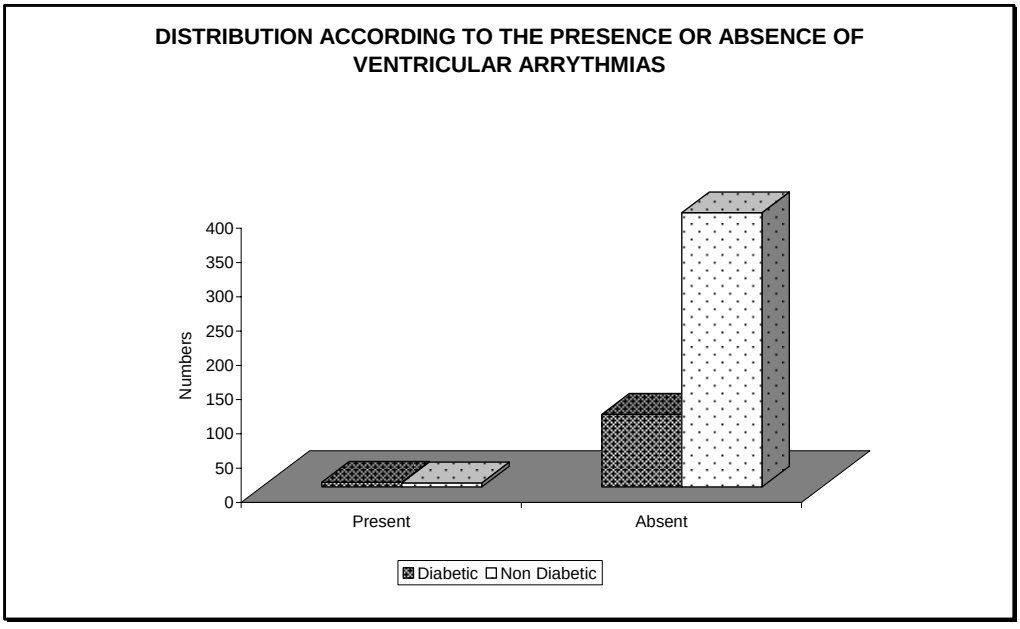


TABLE - 14

DISTRIBUTION ACCORDING TO THE PRESENCE OR ABSENCE OF COMPLETE HEART BLOCK

Group	Complete Heart Block		Total
	Present	Absent	
Diabetic	8	105	113
Non Diabetic	2	404	406

An expected cell value is less than 5.

Fishers exact 2 tailed test used.

P = 0.00012 significant.

Complete Heart blocks were more common in diabetics when compared with non diabetics in myocardial infarction.

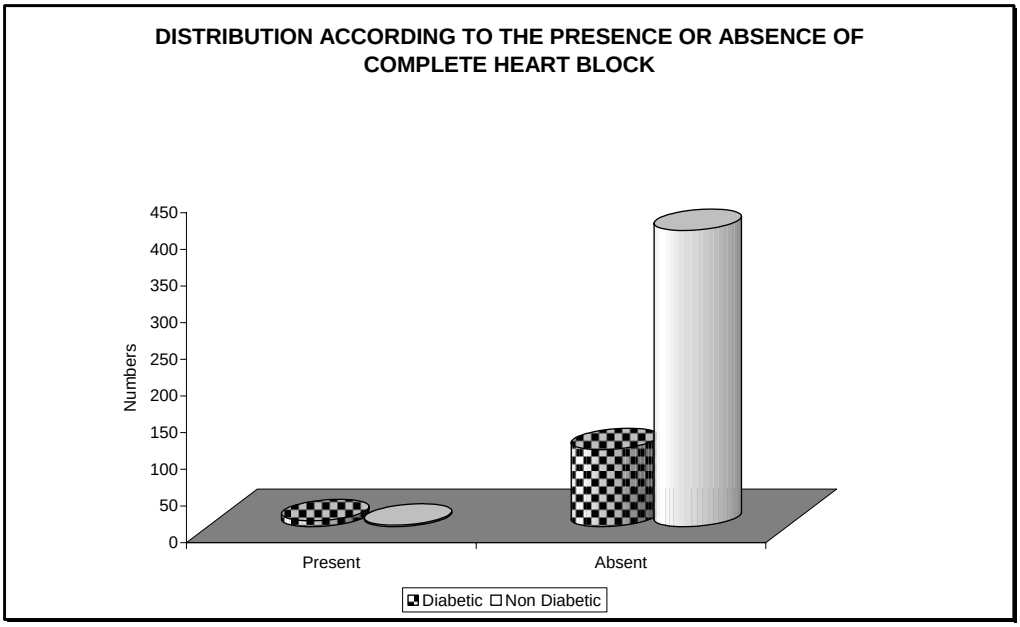


TABLE - 15

DISTRIBUTION ACCORDING TO MORTALITY

Group	Dead	Alive	Total
Diabetic	17	96	113
Non Diabetic	20	38.6	406

$\chi^2 = 13.67$

P value < 0.001 significant

Odds ratio : 3.42

Mortality was 3.42 times more common in the diabetic population compared to non diabetic.

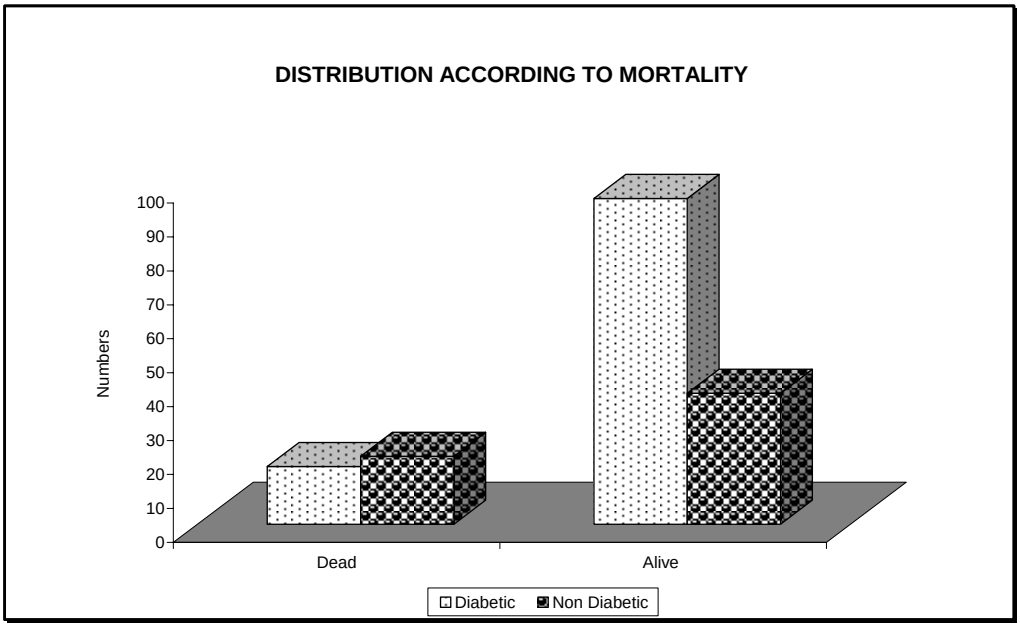
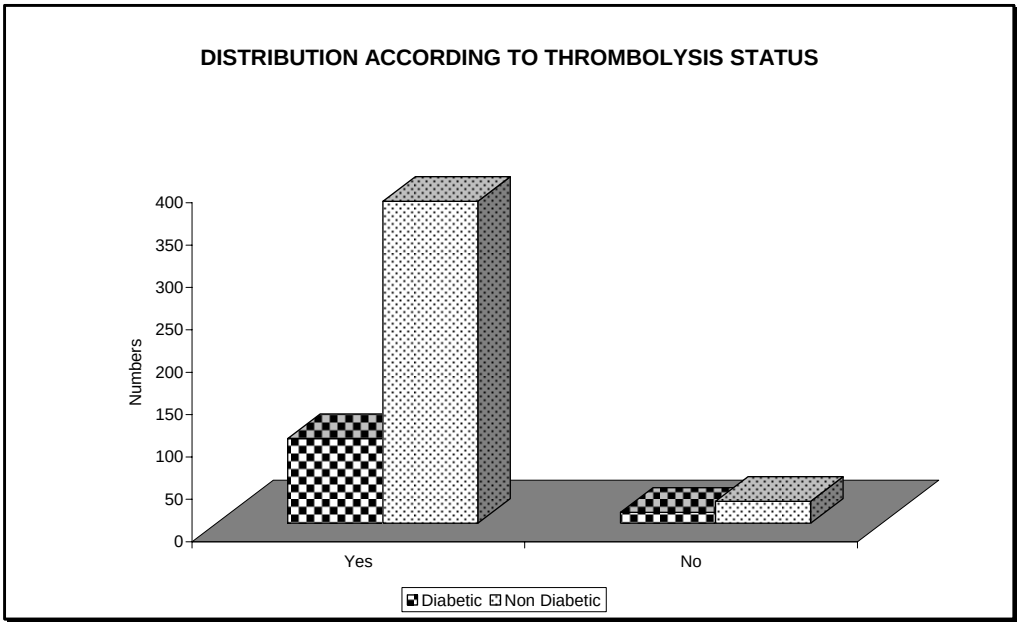


TABLE - 16

DISTRIBUTION ACCORDING TO THROMBOLYSIS STATUS

Group	Thrombolysis		Total
	Yes	No	
Diabetic	100	13	113
Non Diabetic	380	26	406

P value = 0.069 Not significant



Discussion

DISCUSSION

Out of the 519 cases of myocardial infarction in this study, 113 cases were diabetics and 406 cases were non diabetics.

There were 7 cases of myocardial infarction under the age of 30 years in the non diabetic group compared to none in the diabetic group.

Between 31 to 50 years the incidence of myocardial infarction was more common in the non diabetic group compared to diabetic group.

Above the age of 50 years the incidence of myocardial infarction was higher in the diabetic population. Even though there was a slight increase in the age of occurrence of myocardial infarction among the diabetic patients compared to nondiabetics, it was not statically significant.

Percentage of female patients with myocardial infarction in the diabetic group was 29.20% when compared to 16.25% in non diabetic group. This is well in correlation with study of Stone P H et al (14).

42.8% of the patients in the diabetic group had sedentary life style compared to 27.5% of patients in non diabetic group.

It was found that the incidence of painless myocardial infarction were 7.16 times more common in the diabetic group compared to non diabetics ($P<0.001$).

Most of the patients in the diabetic group were having a duration of diabetes less than five years and only 50 percentage of these diabetics were under regular treatment.

Diabetic group showed higher Killip class when compared to non diabetic group. The incidence of congestive cardiac failure was 5.83 times to that of non diabetics ($P<0.001$). The incidence of cardiogenic shock was also higher in the diabetic group. The ejection fraction in diabetic group was very low compared to non diabetics. 8.82% of diabetics had an ejection fraction less than 40% when compared to only 0.51% in non diabetics.

Diabetic patients showed a higher incidence of anterior wall myocardial infarction compared to non diabetics. They had 3.79 times more chance to have anterior wall myocardial infarction compared to non diabetics ($P<0.001$).

The presence of complete heart blocks and life threatening ventricular arrhythmias were also higher in diabetics. The incidence of ventricular arrhythmias in diabetic group was 4.40 times that of non diabetic group.

The mortality due to myocardial infarction among diabetic group was 3.42 times that of non diabetic group.

Conclusion

CONCLUSION

- ✓ In this study, there was no significant difference in the age of occurrence of myocardial infarction among diabetic and nondiabetic group.
- ✓ Women with diabetes lost most of the inherent protection against coronary artery disease when compared to non diabetics.
- ✓ Painless myocardial infarctions were far more common in diabetics compared to nondiabetics.
- ✓ Anterior wall myocardial infarctions were common and ejection fractions were consistently lower among diabetics.
- ✓ Congestive cardiac failure and cardiogenic shock were more common and severe in subjects with diabetes than non diabetics. This was more than to be expected from the size of the infarction.
- ✓ Complications of myocardial infarction like life threatening ventricular arrhythmias and complete heart blocks were far more common among diabetics compared to non diabetics.
- ✓ In hospital mortality due to myocardial infarction in diabetics were three to four times higher that of non diabetics.

Summary

SUMMARY

The present study was conducted over a period two years among patients admitted with ST elevation myocardial infarction in the cardiology department of Kilpauk Medical College. Patients were divided into diabetic and non diabetic groups. With application of inclusion as well as exclusion criteria 113 diabetics and 406 nondiabetics were chosen for the study. Findings of the study are as follows:

1. There was no significant difference in the age of occurrence of myocardial infarction among diabetic and non diabetic group (p value > 0.05).

2. The percentage of females in diabetic group were far more compared to non diabetic group. (diabetic =29.20%, non diabetic =16.25%).

3. The diabetic group had more sedentary life style compared to non diabetics. (diabetic =42.48%, non diabetic =27.40%)

4. Painless myocardial infarctions were 7 times more common in diabetic group. (odds ratio = 7.16) (P value < 0.001).

5. Most of the patients in the diabetic group were detected to have diabetes over the last five years (61.95 %).

6. Diabetic patients with myocardial infarction showed a preponderance to anterior wall compared to non diabetics. (Odds ratio = 3.79).

7. Cardiac failure was about six times more common in the diabetic group (Odds ratio = 5.83) ($P<0.001$).

8. Complications of myocardial infarctions were also common in diabetic group (ventricular arrhythmia- odds ratio = 4.40, complete heart block) ($P<0.001$).

9. Mortality due to myocardial infarction in diabetic group was about four times that of non diabetic group. (odds ratio = 3.42)

10. No significant time difference in receiving thrombolysis was noticed in the two groups ($P>0.05$).

Bibliography

BIBLIOGRAPHY

1. Vergely P. Del' angine de poitrine dans ses rapports ave le diabete *Gaz Hebd Med Chir* **(Paris) (Series 2) 1883 ; 20 : 364-368**.
2. Garcia MJ, McNamara PM, Gordon T, Kannel WB. Morbidity and mortality in diabetics in the Framingham population: sixteen year follow-up study. **Diabetes 1974; 23 : 105–11**.
3. Malmberg K, Ryden L. Myocardial infarction in patients with diabetes mellitus. **Eur Heart J 1988; 9 : 259–64**.
4. Abbud ZA, Shindler DM, Wilson AC, Kostis JB. Effect of diabetes mellitus on short- and long-term mortality rates of patients with acute myocardial infarction: a statewide study. **Am Heart J 1995; 130 : 51– 8**.
5. Partamian JO, Bradley RF. Acute myocardial infarction in 258 cases of diabetes: immediate and five-year survival. **N Engl J Med 1965; 273 : 456–65**.
6. Czyzk A, Krolewski AS, Szablowska S, Alot A, Kopczynski J. Clinical course of myocardial infarction among diabetic patients. **Diabetes Care 1980; 3 : 526–9**.
7. Martin CA, Thompson PL, Armstrong BK, Hobbs MS, de Klerk N.Long-term prognosis after recovery from myocardial infarction: a nine year follow-up of the Perth Coronary Register. **Circulation 1983; 68 : 961–9**.

8. Smith JW, Marcus FI, Serokman R. Prognosis of patients with diabetes mellitus after acute myocardial infarction. **Am J Cardiol 1984; 54 : 718 –21**.

9. Jaffe AS, Spadaro JJ, Schechtman K, Roberts R, Geltman EM, Sobel BE. Increased congestive heart failure after myocardial infarction of modest extent in patients with diabetes mellitus. **Am Heart J 1984; 108 : 31–7**.

10. Rytter L, Troelsen S, Beck-Nielsen H. Prevalence and mortality of acute myocardial infarction in patients with diabetes. **Diabetes Care 1985; 8 : 230–4**.

11. Molstad P, Nustad M. Acute myocardial infarction in diabetic patients. **Acta Med Scand 1987; 222 : 433–7**.

12. Savage MP, Krolewski AS, Kenien GG, Lebeis MP, Christlieb AR, Lewis SM. Acute myocardial infarction in diabetes mellitus and significance of congestive heart failure as a prognostic factor. **Am J Cardiol 1988; 62 : 665–9**.

13. Singer DE, Moulton AW, Nathan DM. Diabetic myocardial infarction: interaction with other preinfarction risk factors. **Diabetes 1989; 38 : 350 –7**.

14. Stone PH, Muller JE, Hartwell T, et al. The effect of diabetes mellitus on prognosis and serial left ventricular function after acute myocardial infarction: contribution of both coronary artery disease and diastolic left ventricular dysfunction to the adverse prognosis. **J. Am Coll Cardiol 1989; 14 : 49 –57**.

15. Granger CB, Califf RM, Young S, et al. Outcome of patients with diabetes mellitus and acute myocardial infarction treated with thrombolytic agents. **J Am Coll Cardiol 1993; 21:920 –5**.

16. Barbash GI, White HD, Modan M, van de Werf F, and the Investigators of the International Tissue Plasminogen / Streptokinase Mortality Trial. Significance of diabetes mellitus in patients with acute myocardial infarction receiving thrombolytic agents. **J Am Coll Cardiol 1993; 22 : 707–13.**

17. Zuanetti G, Latini R, Maggioni AP, Santoro L, Franzosi MG, on behalf of GISSI-2 Investigators. Influence of diabetes on mortality in acute myocardial infarction: data from the GISSI-2 study. **J Am Coll Cardiol 1993; 22 : 1788 –94**.

18. Soler NG, Bennett MA, Lamb P, Pentecost BL, Fitzgerald MG, Malins JM. Coronary care for myocardial infarction in diabetics. **Lancet 1974; 1 : 475–7**.

19. Lomuscio A, Vergani D, Marano L, Castagnone M, Fiorentini C. Effects of glibenclamide on ventricular fibrillation in non–insulin-dependent diabetics with acute myocardial infarction. **Coron Artery Dis 1994; 5 : 767–71**.

20. Luepker RV, Apple FS, Christenson RH, et al : Case definitions for acute coronary disease in epidemiology and clinical research studies. **Circulation 108 : 2543, 2003**.

21. Aplert, JS, Thygesen K. Antman E. et al : Myocardial infarction redefined : A consensus document of The Joint European Society of Cardiology / American College of Cardiology Committee for the redefinition of myocardial infarction. **J Am Coll Cardiol 36 : 959, 2000**.

22. Newby LK, Alpert JS, Ohman EM et al : Changing the diagnosis of acute myocardial infarction : Implications for practice and clinical investigators. **Am Heart J 144 : 957, 2002**.

23. White HD : Things ain't what they used to be : Impact of a new definition of myocardial infarction. **Am Heart J 144 : 933, 2002**.

24. Fuster V. Corti R, Fayad ZA, et al: Integration of vascular biology and magnetic resonance imaging in the understanding of atherothrombosis and acute coronary syndromes. **J Thromb Haemost 1:1410,2003**.

25. Libby P: Current concepts of the pathogenesis of the acute coronary syndromes. **Circulation 104:365,2001**.

26. Ardissino D, Merlini PA, Ariens R. et al: Tissue – factor antigen and activity in human coronary atherosclerotic plaques. **Lancet 349-769, 1997**.

27. Malek AM, Alper SL, Izumo S: Hemodynamic shear stress and its role in atherosclerosis. **JAMA 282:2035, 1999**.

28. Kloner RA, Leor J: Natural disaster plus wake-up time .A deadly combination of triggers. **Am Heart J 137:799, 1999**.

29. Barrett-Connor EL, Cohn BA, Wingard DC, et al. Why is diabetes mellitus a stronger risk factor for fatal ischemic heart disease in women than in men? The Rancho Bernardo Study (published erratum appears in *JAMA* 1991; 265 : 3249)**. JAMA 1991; 265 ; 627-631**.

30. Gu K, Cowie CC, Harris MI. Mortality in adults with and without diabetes in a national cohort of the U.S. population, 1971-1993. **Diabetes Care 1998 ; 21 : 1138-145**.

31. Krolewski, AS, Kosinski EJ, Warram JH, et al. Magnitude and determinants of coronary artery disease in juvenile-onset, insulin-dependent diabetes mellitus, **Am J Cardiol 1987 : 750-755**.

32. Krolewski, AS, Warram JH, Rand LI, et al. Epidemiologic approach to the etiology of type I diabetes mellitus and its complications. **N Engl J Med 1987; 317 : 1390-1398**.

33. Bale GS, Entmacher PS. Estimated life expectancy of diabetics. **Diabetes 1877; 26: 434-438**.

34. Haffner SM, Lehtol S, Ronnemaa T, et al. Mortality from coronary heart disease in subjects with type 2 diabetes and in nondiabetic subjects with and without prior myocardial infarction. **N Engl J Med 1998; 339:229 – 234**.

35. Mukamal KJ, Nesto RW, Cohen MC, et al. Impact of diabetes on long-term survival after acute myocardial infarction: comparability or risk with prior myocardial infarction. **Diabetes Care 2001; 24: 1422-1427**.

36. Malmberg K, Kusuf S, Gerstein HC, et al. Impact of diabetes on long-term prognosis in patients with unstable angina and non-Qwave myocardial infarction: results of the OASIS (Organization to Assess Strategies for Ischemic Syndromes) Registry. **Circulation2000; 102: 1014- 1019**.

37. Gu K, Cowie Cc, Harris MI. Diabetes and decline in heart disease mortality in U.S adults. **JAMA 1999; 281:1291-1297**.

38. Donahue RP, Orchard, TJ. Diabetes mellitus and macrovascular. complications. An epidemiological perspective. **Diabetes Care 1992; 15 : 1141-1155**.

39. Maser RE, Wolfson SK, Jr, Ellis D, et al. Cardiovascular disease and arterial calcification in insulin-dependent diabetes mellitus: interrelations and risk factor profiles. Pittsburgh Epidemiology of Diabetes Complications Study. **V. Arterioseler Thromb 1991; 11: 958-965.**

40. Jensen T, Borch-Johnsen K, Kofoed-Enevoldsen A, et al. Coronary heart disease in young type 1 (insulin-dependent) diabetic patients with and without diabetic nephropathy; incidence and risk factors. **Diabetologin 1987; 30 : 144-148**.

41. Kannel WB, McGee DL. Diabetes and cardiovascular disease. The Framingham Study, **JAMA 1979; 241 : 2035-2038**.

42. Jarrett RJ, McCartney P,Keen H. The Bedford survey : ten year mortality rates in newly diagnosed diabetics, borderline diabetics and normoglycaemic controls and risk indices for

coronary heart disease in borderline diabetics. **Diabetologia 1982 ; 22 : 79-84**.

43. Jarrett RJ, Shipley MJ. Type 2 (non-insulin-dependent)diabetes mellitus and cardiovascular disease – putative association via common antecedents; further evidence from the Whitehall study. **Diabetologia 1988 ; 31 : 737-740**.

44. Fontbonne A, Eschwege E, Cambient F., et al. Hypertriglycerdaemia as a risk factor of coronary heart disease mortality in subjects with impaired glucose tolerance or diabetes. Results from the 11-year follow-up of the Paris Prospective Study. **Diabetologia 1989; 32 : 300-304**.

45. Nathan DM. Long-term complications of diabetes mellitus. **N Engl J Med 1993; 328 : 1676-1685**.

46. Barrett-Connor E, Wingard DL. Sex differential in ischemic heart disease mortality in diabetics : a prospective population – based study. **Am J Epidemiol 1983; 118 : 489-496**.

47. Reaven G. Syndrome X : 10 yers after. **Drugs 1999; 58 (Suppl (1) : 19-20**.

48. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP). Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adults Treatment Panel III). **JAMA 2001 : 285 : 2486-2497**.

49. Ferrannini E, Buzzigoli G, Bonadonna R, et al. Insulin resistance in essential hypertension . **N Engl J Med 1987; 317: 350-357**.

50. Zavaroni I, Bonora E, Pagliara M, et al. risk factors for coronary artery disease in healthy person with hyperinsulinemia and normal glucose tolerance. **N Engl J Med 1989; 320: 702-706**.

51. Larsson B, Svardsudd K, Welin L, et al. abdominal adipose tissue distribution, obesity, and risk of cardiovascular disease and death : 13 year follow up of participants in the study of men born in 1913. **BMJ (Clin Res Ed) 1984 ; 288 : 1401-1404**.

52. Laakso M, Barrett – Connor E. Asymptomatic hyperglycemia is associated with lipid and lipoprotein changes favouring atherosclerosis. **Arteriosclerosis 1989 ; 9 : 665-672**.

53. Laws A, King AC, Haskell WL, et al. Relation of fasting plasma insulin concentration to high density lipoprotein cholesterol and triglyceride concentrations in men. **Arterioscler Thromb 1991 ;11: 1636-1642**.

54. Modan M, Halkin H, Lusky A, et al. Hyperinsulinemia is characterized by jointly disturbed plasma VLDL, LDL, and HDL levels. A population –based study. **Arteriosclerosis 1988 ; 8: 227-236**.

55. Peiris AN, Sothmany S, Hoffman RG, et al. Adiposity, fat distribution, and cardiovascular risk. **Ann Intern Med 1989; 110: 867-872**.

56. Howard, G, O' Leary DH, Zaccaro D, et al. Insulin sensitivity and atherosclerosis. The Insulin Resistance Atherosclerosis Study (IRAS) Investigators. **Circulation 1996; 93: 1809-1817**.

57. Laakso M, Sarlund H, Salonen R, et al. Asymptomatic atherosclerosis and insulin resistance. **Arterioscler, Thromb 1991 ; 11: 1068-1076**.

58. Agewall S, Wikstrand J, Dahlof C, et al. Urinary albumin excretion is associated with the intima-media thickness of the caroid artery in hypertensive males with non-insulin-dependent diabetes mellitus. **J Hypertens 1995;13:463-469**.

59. Bonora E, Kiechl S, Williet J, et al. Prevalence of insulin resistance in metabolic disorders; the Bruneck Study. **Diabetes 1998; 47: 1643-1649**.

60. Fuller JH, Shipley MJ, Rose G, et al. coronary – heart – disease risk and impaired glucose tolerance. The Whitehall Study. **Lancet 1980;1: 1373-1376**.

61. Morrish NJ, Stevens LK, Head J, et al. A prospective study of mortality among middle-aged diabetic patients (the London Cohort of the WHO Multinational Study of Vascular Disease in Diabetes) II : Associated risk factors (published erratum

appears in *Diabetologia* 1991; 34:287). **Diabetologia 1990;3: 542-548**.

62. Uusitupa M, Siitoneno, Pyorala K, et al. The relationship of cardiovascular risk factors to the prevalence of coronary heart disease in newly diagnosed type 2 (non-insulin-dependent) diabetes. **Diabetologia 1985; 28 : 653-659**.
63. Head J, Fuller JH, International variations in mortality among diabetic patients: the WHO Multinational Study of Vascular Disease in Diabetics. **Diabetologia 1990; 33:477-481**.
64. Despres JP, Lamarch B, Mauriege P*, et al*. Hyperinsulinemia as an independent risk factor for ischemic heart disease. **N Engl J Med 1996; 334:952-957**.
65. Andersson DK, Svardsudd K. Long – term glycemic control relates to mortality in type II diabetes. **Diabetes Care 1995; 18: 1534-1543**.
66. Wei M, Gaskill SP, Haffner SM , et al. Effects of diabetes and level of glycemia on all – cause and cardiovascular mortality. The San Antonio Heart Study. **Diabetes Care 1998; 21:1167-1172**.
67. Klein R, Klein BE, Moss SE. The Wisconsin epidemiologic study of Diabetic Retinopathy. XVI. The relationship of C-peptide to the incidence and progression of diabetic retinopathy. **Diabetes 1995; 44: 796-801**.

68. Wingard DL, Barrett- Connor EL, Scheidt – Nave C, et al. Prevalence of cardiovascular and renal complications in older adults with normal or impaired glucose tolerance or NIDDM. A population- based study. **Diabetes Care 1993; 16:1022-1025**.

69. Kuusisto J, Mykkanen L, Pyorala K, et al. Non – insulin-dependent diabetes and its metabolic control are important predictors of stroke in elderly subjects. **Stroke 1994; 25: 1157-1164**.

70. Rodriguez BL, Lau N, Burchifiel CM, et al. Glucose introlerance and 23-years risk of coronary heart disease and total mortality: the Honolulu Heart Program. **Diabetes Care 1999; 22:1262 – 1265**.

71. Folsom AR,Eckfeldt JH,Weitzman S , et al. Relation of carotid artery wall thickness to diabetes mellitus, fasting blood glucose and insulin, body size, and physical activity. Atherosclerosis Risk in Communities (ARIC) Study Investigators. **Stroke 1994;25:66-73**.

72. Singer DE, Nathan DM, Anderson KM, et al. Association of HbA1c with prevalent cardiovascular disease in the original cohort of the Framingham Heart Study. **Diabetes 1992; 41: 202 – 208**.

73. Kuusisto J, Kykkanen L, Pyorala K, et al. NIDDM and its metabolic control predict coronary heart disease in elderly subjects. **Diabetes 1994; 43: 960-967**.

74. Mattock MB, Morrish NJ, Viberti G, et al. Prospective study of microalbuminuria as predictor of mortality in NIDDM. **Diabetes 1992; 41: 736-741**.

75. Neil a, Hawkins M, Potok M, et al. A prospective population – based study of microalbuminuria as a predictor of mortality in NIDDM. **Diabetes Care 1993; 16: 996 – 1003**.

76. Ducimetiere P, Eschwege, T, Papoz L, et al. Relationship of plasma insulin levels to the incidence of myocardial infarction and coronary heart disease mortality in a middle – aged population. **Diabetologia 1980; 19:205-210**.

77. Pyorala K. Relationship of glucose tolerance and plasma insulin to the incidence of coronary heart disease: results from two population studies in Finland. **Diabetes Care 1979; 2:131-141**.

78. Welborn TA, Wearne K. Coronary heart disease incidence and cardiovascular mortality in Busselton with reference to glucose and insulin concentration. **Diabetes Care 1979; 2 : 154-160**.

79. Stout RW, Insulin and atheroma: 20 yr perspective. **Diabetes Care 1990; 13: 631-654**.

80. Gowri MS,van der Westhuyzen DR,Bridges S R ,et al. Decreased protection by HDL from poorly controlled type 2 diabetic subjects against LDL oxidation may be due to abnormal composition of HDL. **Arterioscler Thromb vasc biol 1999;19:2226-2233**.

81. Tsai E C,Hirch I B,Brunzell J D,et al.Reduced plasma peroxy radical trapping capacity and increased susceptibility of LDL to oxidation in poorly controlled IDDM. **Diabetes 1994;43:1010-1014 82**.

82. Baynes JW, Thorpe SR, Role of oxidative stress in diabetic complications : a new perspective on an old paradigm. **Diabetes 1999;48:1-9**.

83. Ceriello A, Quatraro A, Caretta F, et al. Evidence for a possible role of oxygen free radicals in the abnormal arterial vasomotor function in insulin dependent diabetes. **Diabetes Metab 1990;16: 318-322**.

84. Kilhovd BK, Berg TJ, Birekeland KJ, et al. Serum levels of advanced glycation end products are increased in patients with type 2 diabetes and coronary heart disease. **Diabetes Care 1999;22: 1543-1548**.

85. Baynes JW. Role of oxidative stress in development of complications in diabetes. **Diabetes 1991; 40: 405-412**.

86. King GL, Shiba T, Oliver J, et al. Cellular and molecular abnormalities in the vascular endothelium of diabetes mellitus. **Annu Rev Med 1994;45: 179-188**.

87. Gabbay KH. The sorbitol pathway and the complications of diabetes. **N Engl J Med 1973; 288: 831-836**.

88. Ahmed MU, Thorpe SR, Baynes JW. Identification of *N*-epsilon-carboxymethyllysine as a degradation product of

fructoselysine in glycated protein. **J Biol Chem 1986; 261: 4889-4894**.

89. Sell DR, Monnier VM. Structure elucidation of a senescence cross-link from human extracellular matrix. Implication of pentoses in the aging process. **J Biol Chem 1989; 264: 21597-21602**.

90. Baron AD. Insulin and the vasculature-old actors, new roles. **J Invest Med 1996; 44: 406-412**.

91. Moncada S. Eighth Gaddum Memorial Lecture, University of London Institute of Education, December 1980. Biological importance of prostacyclin. **Br J Pharmacol 1982; 76: 3-31**.

92. Bucala R, Makita Z, Koschinsky T, et al. Lipid advanced glycosylation: pathway for lipid oxidation in vivo. **Proc Nalt Acad Sci U S A 1993; 90: 6434- 6438**.

93. Brown AS, Hong AS, De Belder A, et al. Megakaryocyte ploidy and platelet changes in human diabetes and atherosclerosis. **Arterioscler Thromb Vasc Biol 1997: 17; 802-807**.

94. Winocour PD, Watala C, Kinghlough – Rathbone RL. Membrane fluidity is related to the extent of glycation of protein, but not to alterations in the cholesterol to phospholipid molar ratio in isolated platelet membranes from diabetic and control subjects. **Thromb Haemost 1992; 67: 567-571**.

95. Davi G, Catalano I, Averna M, et al Thromboxane biosynthesis and platelet function in type II diabetes mellitus. **N Engl J Med 1990; 322: 1769-1771**.

96. Ishii H, Umeda F, Nawata H. Platelet function in diabetes mellitus. **Diabetes Metab Rev 1992; 8: 53-66**.

97. Hendra T, Betteridge DJ. Platelet function, platelet prostanoids and vascular prostacyclin in diabetes mellitus. **Prosaglandins Leuket Essent Fatty Acids 1989; 35: 197-212**.

98. Menys VC, Bhatnagar D, Mackness Ml, et al. Spontaneous platelet aggregation in whole blood is increased in non-insulin-dependent diabetes mellitus and in female but not male patients with primary dyslipidemia. **Atherosclerosis 1995; 112: 115-122**.

99. Kannel WB, D'Agostino RB, Wilson PW, et al. Diabetes, fibrinogen, and risk of cardiovascular disease : the Framingham experience. **Am Heart J 1990; 120: 672-676**.

100. Lufkin EG, Fass DN, O'Fallon WM, et al. Increased von Willebrand factor in diabetes mellitus. **Metabolism 1979; 28: 63-66**.

101. Kannel WB, Wolf PA, Castelli WP, et al. Fibrinogen and risk of cardiovascular disease. The Framingham Study. **JAMA 1987; 258: 1183-1186**.

102. Eliasson M, Roder ME, Dinesen B, et al. Proinsulin, intact insulin, and fibrinolytic variables and fibrinogen in healthy

subjects. A population study. **Diabetes Care 1997: 20; 1252-1255**.

103. Ceriello A, Dello Russo P, Zuccotti C, et al. Decreased antithrombin III activity in diabetes may be due to non-enzymatic glycosylation – a preliminary report. **Thromb Haemost 1983; 50: 633-634**.
104. Brownlee M, Vlassara H, Cerami A. Inhibition of heparin-catalyzed human antithrombin III activity by non-enzymatic glycosylation. Possible role in fibrin deposition in diabetes. **Diabetes 1984; 33: 532-535**.
105. Ceriello A, Giugliano D, Quatraro A, et al. Daily rapid blood glucose variations may condition antithrombin III biologic activity but not its plasma concentration in insulin-dependent diabetes. A possible role for labile non-enzymatic glycation. **Diabetes Metab 1987:13; 16-9**.
106. Ceriello A, Quatraro A, Dello Russo P, et al. Protein C deficiency in insulin – dependent diabetes : a hyperglycemia – related phenomenon. **Thromb Haemost 1990; 64: 104-107**.
107. Auwerx J, Bouillon R, Collen D, et al. Tissue-type plasminogen activator antigen and plasmingen activator inhibitor in diabetes mellitus. **Arleriosclerosis 1988; 8: 68-72**.
108. McGill JB, Schneider DJ, Arfken CL, et al. Factors responsible for impaired fibrinolysis in obese subjects and NIDDM patients. **Diabetes 1994; 43: 104-109**.

109. Nordt TK, Schneider DJ, Sobel BE. Augmentation of the synthesis of plasminogen activator inhibitor type-1 by precursors of insulin. A potential risk factor for vascular disease. **Circulation 1994; 89: 321-330**.

110. Furchgott RF, Zawadzki JV, The obligatory role of endothelial cells in the relaxation of arterial smooth muscle by acetylchoine. **Nature 1980; 288: 373-376**.

111. Palmer RM, Ferrige AG; Moncada S. Nitric oxide release, accounts for the biological activity of endothelium- derived relaxing factor. **Nature 1987; 327: 524-526**.

112. Ignarro LJ, Buga GM, Wood Ks, et al. Endothelium-derived relaxing factor produced and released from artery and vein is nitric oxide. **Proc Natl Acad Sci U S A 1987; 84: 9265-9926**.

113. Dinerman JL, Lowenstein CJ, Snyder SH. Molecular mechanisms of nitric oxide regulation. Potential relevance to cardiovascular disease. **Circ Res 1993; 73: 217-222**.

114. Lincoln TM, Cornwell TL, Taylor AE. cGMP – dependent protein kinase mediates the reduction of Ca^{2+} by cAMP in vascular smooth muscle cells. **Am J physiol 1990; 258: (pt 1) : C399-C407**.

115. Collins P, Chappell SP, Griffith TM, et al. Differences in basal endothelium derived relaxing factor activity in different artery types. **J Cardiovasc Pharmacol 1986; 8: 1158-1162**.

116. Johnstone MT, Creager SJ, Scales KM, et al. Impaired endothelium dependent vasodilation in patients with insulin-dependent diabetes mellitus. **Circulation 1993; 88: 2510-2516**.

117. McVeigh GE, Brennan GM, Roddy MA, et al. Imparied endothelium – dependent and independent vasodilation in patients with type 2 (non-insulin-dependent) diabetes mellitus. **Diabetologia 1992:35; 771-776**.

118. Williams SB, Cusco JA, Roddy MA, et al. Impaired nitric oxide – mediated vasodilation in patients with non-insulin-dependent diabetes mellitus. **J Am Coll Cardiol 1996; 27: 527-574**.

119. Steinberg HO, Chaker H, Leaming R, et al. Obesity / insulin resistance is associated with endothelial dysfunction. Implications for the syndrome of insulin resistance. **J Clin Invest 1996; 97: 2601-2610**.

120. Ting HH, Timimi FK, Boles KS, et al. Vitamin C. Improves endothelium dependent vasodilation in patients with non-insulin-dependent diabetes mellitus. **J Clin Invest 1996; 97: 22-28**.

121. Timimi FK, Ting H, Haley EA, et al. Vitamin C improves endothelium dependent vasodilation in patients with insulin-dependent diabetes mellitus. **J Am Coll Cardiol 1998; 31: 552-557**.

122. Van Etten RW, De Koning EJ, Honing MI, et al. Intensive lipid lowering by statin therapy does not improve vasoreactivity in patients with type 2 diabetes. **Arterioscler Thromb Vasc Biol 2002; 22: 799-804**.

123. Bradley RF, Schonfeld A. Diminished pain in diabetic patient with acute myocardial infarction. **Geriatrics 1962; 17: 322-326**.

124. Margolis JR, Kannel WS, Feinleib M, et al. Clinical feature of unrecognized myocardial infarction – silent and asymptomatic. Eighteen year follow-up, the Framingham Study. **Am J Cardiol 1973; 32: 1-7**.

125. Soler NG, Bennett MA, Pentecost BL, et al. Myocardial infarction in diabetics. **Q J Med 1975; 44: 125-132**.

126. Marchant B, Umanchandran V, Stevenson R, et al. Silent myocardial ischemia: role of subclinical neuropathy in patients with and without diabetes. **J Am Coll Cardiol 1993; 22: 1433-1437**.

127. Hume L, Oakley GD, Boulton AJ et al. Asymptomatic myocardial ischemia in diabetes and its relationship to diabetic neuropathy : an exercise electrocardiography study in middle-aged diabetic men. **Diabetes Care 1986; 9: 384-388**.

128. O'Sullivan J, Conroy RM, MacDonald K, et al. Silent ischaemia in diabetic men with autonomic neuropathy. **Br Heart J 1991:66; 313-315**.

129. Nesto RW, Phillips RT, Kett KG, et al. Angina and exertional myocardial ischemia in diabetic and nondiabetic patients : assessment by exercise thallium scintigraphy (published erratum appears in *Ann Intern Med* 1988; 108: 646). **Ann Intern Med 1988; 108: 170-175**.

130. Abenavoli T, Rubler S, Fisher VJ, et al. Exercise testing with myocardial scintigraphy in asymptomatic diabetic males. **Circulation 1981; 63: 54-64**.

131. Langer A, Freeman MR, Josse RG, et al. Detection of silent ischemia : age, diabetes mellitus, previous myocardial infarction and prognosis. **Amj Cardiol 1991; 67: 1073-1078**.

132. Milan Study on Atherosclerosis and Diabetes (MiSAD) Group. Prevalence of unrecognized silent myocardial ischemia and its association with atherosclerotic risk factors in noninsulin-dependent diabetes mellitus. **Am J Cardiol 1997; 79: 134-139**.

133. Callaham PR, Froelicher VF, Klein J, et al. Exercise – induced silent ischemia age, diabetes mellitus, previous myocardial infarction and prognosis. **J Am Coll Cardiol 1989; 14: 1175-1180**.

134. Caracciolo EA, Chaitman BR, Forman SA, et al. Diabetics with coronary disease have a prevalence of asymptomatic ischemia during exercise treadmill testing and ambulatory ischemia monitoring similar to that of nondiabetic patients. An ACIP database study. ACIP Investigators. Asymptomatic

Cardiac Ischemia Pilot Investigators. **Circulations 1996; 93: 2097-2015**.

135. Faerman I, Faccio E, Milei J, et al. Autonomic neuropathy and painless myocardial infarction in diabetic patients. Histologic evidence of their relationship. **Diabetes 1977; 26: 1147-1158.**

136. Ambepityia G, Kopelman PG, Ingram D, et al. Exertional myocardial ischemia in diabetes : a quantitative analysis of anginal perceptual threshold and the influence of autonomic function. **J Am Coll Cardiol 1990;15: 72-77**.

137. Granger CB, Califf RM, Young S, et al. Outcome of patients with diabetes mellitus and acute myocardial infarction treated with thrombolytic agents. The Thrombolysis and Angioplasty in Myocardial Infarction (TAMI) Study Group. **J Am Coll Cardiol 1993; 21: 920-925**.

138. Jaffe AS, Spadaro JJ, Schechtman K, et al. Increased congestive heart failure after myocardial infarctin of modest extent in patients with diabetes mellitus. **Am Heart J 1984; 108: 31-37**.

139. Savage MP, Krolewski AS, Kenien GG, et al. Acute myocardial infarction in diabetes mellitus and significance of congestive heart failure as a prognostic factor. **Am J Cardiol 1988; 62 (10 pt 1): 665-669**.

140. Stone PH, Muller JE, Hartwell T, et al. The effect of diabetes mellitus on prognosis and serial left ventricular function after

acute myocardial infarction : contribution of both coronary disease and diastolic left ventricular dysfunction to the adverse prognosis. The MILIS Study Group. **J Am Coll Cardiol 1989; 14: 49-57**.

141. Mak KH, Molitemo DJ, Granger CB, et al. Influence of diabetes mellitus on clinical outcome in the thrombolytic era of acute myocardial infarction. GUSTO-I Investigators, Global Utilization of Streptokinase and Tissue plasminogen Activator for Occulded Coronary Arteries. **J Am Coll Cardiol 1997; 30: 171-179**.
142. Barbash GI, White HD, Modan M, et al. Significance of diabetes mellitus in patients with acute myocardial infarction receiving thrombolytic therapy Investigators of the International Tissue Plasminogen Activator/ Streptokinase Mortality Trial. **J Am Coll Cardiol 1993; 22: 707-713**.
143. Lee KL, Woodlief LH, Topol EJ, et al. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction. Results from an international trial of 41,021 patients GUSTO-I Investigators. **Circulation 1995; 91: 1659-1668**.
144. Zuanetti G, Latini R, Maggioni A, et al. Influence of diabetes on mortality in acute myocardial infarction : data from the GISSI-2 study. **J Am Coll Cardiol 1993; 22: 1788-1794**.
145. Holmes DR Jr, Berger PB, Hochman JS, et al. Cardiogenic shock in patients with acute ischemic syndromes with and

without ST-segment elevation. **Circulation 1999; 100: 2067-2073**.

146. Orlander PR, Goff DC, Morrissey M, et al. The relation of diabetes to the severity of acute myocardial infarction and post – myocardial infarcion survival in Mexican – Americans and non-Hispanic whites. The Corpus Christi Heart Project. **Diabetes 1994; 43: 897-902**.
147. Lehto S, Pyorala K, Miettinen H, et al. Myocardial infarct size and mortality in patients with non-insulin-dependent diabetes mellitus. **J Inten Med 1994; 236: 291-297**.
148. Ulvenstam G Aberg A, Bergstrand R, et al. Long-term prognosis after myocardial infarction in men with diabetes. **Diabetes 1985; 34: 787-792**.
149. Iwasaka T, Takahashi N, Nakamura S, et al. Residual left ventricular pump function after acute myocardial infarction in NIDDM patients. **Diabetes Care 1992; 15: 1522-1526**.
150. Aronson D, Rayfield EJ, Chesebro JH. Mechanisms determining course and outcome of diabetic patients who have had acute myocardial infarction. **Am Intern Med 1997; 126: 296-306**.
151. Fava S, Azzopardi J, Muscat HA, et al. Factors that influences outcome in diabetes subjects with myocardial infarction. **Diabetes Care 1993; 16: 1615-1618**.

152. Gwilt DJ, Petri M, Lewis PW, et al. Myocardial infarct size and mortality in diabetic patients. **Br Heart J1985; 54: 466-472**.

153. Zarich SW, Arbuckle BB, Cohen LR, et al. Diastolic abnormalities in young asymptomatic diabetic patients assessed by pulsed Doppler echocardiography. **J Am Coll Cardiol 1988; 12: 114-120**.

154. Takahashi N, Iwasaka T, Sugiura T, et al. Left ventricular regional function after acute anterior myocardial infarction in diabetic patients. **Diabetes Care 1989; 12: 630-635**.

155. The GUSTO Angiographic Investigators. The effects of tissue plasminogen activator, streptokinase, or both on coronary-artery patency, ventricular function, and survival after acute myocardial infarction. **N Engl J Med 1993; 329: 1615-1622**.

156. Abaci A, Oguzhan A, Kahraman S, et al. Effect of diabetes mellitus on formation of coronary collateral vessels. **Circulation 1999; 99: 2239-2242**.

157. Herlitz J, Malmberg K, Karlson BW, et al. Mortality and morbidity during a five-year follow-up of diabetics with myocardial infarction. **Acta Med Scand 1988; 224: 31-38**.

158. Capone RJ, Pawitan Y, el-Sherif N, et al. Events in the cardiac arrhythmia suppression trial : baseline predictors of mortality in placebo-treated patients. **J Am Coll Cardiol 1991; 18: 1434-1438**.

159. Gilpin E, Ricou F, Dittrich H, et al. Factors associated with recurrent myocardial infarction within one year after acute myocardial infarction. **Am Heart J 1991; 121(2 Pt 1) : 457-465**.

160. Taylor GJ, Moses HW, Katholi RE, et al. Six year survival after coronary thrombolysis and early revascularization for acute myocardial infarction. **Am J Cardiol 1992; 70: 26-30**.

161. Garcia MJ, McNamara PM, Gordon T, et al. Morbidity and mortality in diabetics in the Framingham population. Sixteen-year follow-up study. **Diabetes 1974; 23: 105-111**.

162. Jones SL, Close CF, Mattock MB, et al. Plasma lipid and coagulation factor concentrations in insulin dependent diabetics with microalbuminuria. **BMJ 1989; 298: 487-490**.

163. Hamby RI, Zoneraich S, Sherman L. Diabetic cardiomyopathy. **JAMA 1974; 229: 1749-1754**.

164. Litwin SE, Grossman W. Diastolic dysfunction as a cause of heart failure. **J Am Coll Cardiol 1993; 22 (Suppl 4A) : 49A-55A**.

165. Timmis AD. Diabetic heart disease : clinical considerations. **Heart 2001; 85: 463-469**.

166. Roffi M, Moliterno DJ, Meler B, et al. Impact of different platelet glycoprotein IIb/IIIa receptor inhibitors among diabetic patients undergoing percutaneous coronary intervention: Do Tirofiban and Reopro Give Similar Efficacy

Outcomes Trial (TARGET) 1-year follow-up. **Circulation 2002; 105: 2730-2736**.

167. Alexander RW,Ryan TJ,Pratt CM:ST-Segment Elevation Myocardial Infarction, in O'Rourke RA (ed): Hurst's The Heart Manual of Cardiology,11th ed., Boston, McGraw Hill, **2005, 1283**.
168. Schamroth L: Myocardial Infarction, in Schamroth C (ed): An introduction to electrocardiography, 7th ed. Oxford, Blackwell Science Ltd., **2003, pp 142 – 148**.
169. Antman EM,Braunwald E:ST-segment Elevation Myocardial Infarction, in Kasper DL (ed):Harrison's Principles of Internal Medicine. 16th ed. New York, McGraw Hill, **2005, 1456**.
170. Schamroth L: Ventricular Rhythms: An introduction to electrocardiography, 7th ed. Oxford, Blackwell Science Ltd., **2003, 357-366**.
171. Josephson ME, Zimetbaum P: Tachyarrhythmias, in Kasper DL (ed):Harrison's Principles of Internal Medicine. 16th ed. New York, McGraw Hill, **2005,1351**.
172. Schamroth L: Atrioventricular Block: An introduction to electrocardiography, 7th ed. Oxford, Blackwell Science Ltd., **2003, 379**.
173. Powers AC: Diabetes Mellitus, in Kasper DL (ed):Harrison's Principles of Internal Medicine. 16th ed. New York, McGraw Hill, **2005, 2153-2154**.

ABBREVIATIONS

ADP	-	Adenosine Diposphate
AGEs	-	Advancedglycation End Products
ALMI	-	Anterolateral MI
ASMI	-	Anteroseptal MI
AWMI	-	Extensive Anterior Wall MI
BP	-	Blood Pressure
CAD	-	Coronary Artery Disease
CK-MB	-	Creatine Kinase - MB
CML	-	Carboxymethyllysine
CVS	-	Cardiovascular System
ECG	-	Electrocardiography
EDRF	-	Endothelium Derived Relaxing Factor
EF	-	Ejection Fraction
F	-	Female
FBS	-	Fasting Blood Sugar
GOD	-	Glucose Oxidase
HDL	-	High Density Lipoprotein

IWMI	-	Inferior Wall MI
LDL	-	Low Density Lipoprotein
M	-	Male
M I	-	Myocardial Infarction
ML	-	Manual Labourer
No.	-	Number
PAI-1	-	Plasminogen Activator Inhibitor-1
POD	-	Peroxidase
PWMI	-	Posterior Wall MI
RBS	-	Random Blood Sugar
S	-	Sedentary
STEMI	-	ST-Elevation Myocardial Infarction
t-PA	-	tissue Plasminogen Activator
VLDL	-	Very Low Density Lipoprotein

PROFORMA

NAME :

AGE :

SEX : M/F DOA :

OCCUPATION :- M L/ S DOD :

SYMPTOMS

CHIEF PRESENTING COMPLAINT:

- CHEST PAIN
- BREATHLESSNESS
- PALPITATION
- SYNCOPE
- NONE OF THE ABOVE

DIABETIC : Y/N

DURATION : <5yrs
5 – 10 yrs
10 – 15 yrs
>15 yrs

TREATMENT:- Regular / Irregular

CLINICAL EXAMINATION

- PULSE
- BP
- KILLIP Class.
- CVS

INVESTIGATION

FBS

RBS

ECG :- Extensive Anterior Wall (AWMI)

Anteroseptal Wall (ASMI)

Anterolateral Wall (ALMI)

Inferior Wall (IWMI)

ECHO

Wall involved -

Ejection Fraction -

COMPLICATION

CARDIAC FAILURE – Yes / No

LIFE THREATENING ARRYTHMIA – Yes / No

PERICARDITIS – Yes / No

THROMBOEMBOLISM – Yes / No

PAPILLARY MUSCLE DYSFN. – Yes / No

VENTRICULAR SEPTAL RUPTURE – Yes / No

DEATH:- Yes/ No

MASTER CHART

S.No	Age	Sex	Occ.	P Com.	DM	Dur.	Rx	P	K C	FBS	RBS	ECG	EF	Comp	Mort
1	65	F	S	CP	-	-	-	82	II	78	120	ASMI	60	CCF	-
2	62	F	S	CP	-	-	-	60	I	90	110	IWMI	60	-	-
3	61	F	S	CP	-	-	-	88	I	80	90	ALMI	6O	-	-
4	35	M	M	CP	-	-	-	80	I	74	86	ASMI	54	-	-
5	46	M	M	CP	-	-	-	76	I	80	120	ASMI	60	-	-
6	44	M	S	CP	-	-	-	80	I	80	100	ASMI	60	-	-
7	60	M	M	CP	-	-	-	80	I	64	124	AWMI	50	-	-
8	48	M	M	CP	-	-	-	90	I	80	125	ASMI	60	-	-
9	70	M	S	CP	-	-	-	84	I	110	136	ASMI	60	-	-
10	48	M	M	CP	-	-	-	90	I	70	100	ALMI	60	-	-
11	59	F	M	CP	-	-	-	80	I	74	128	IWMI	62	-	-
12	54	M	M	CP	-	-	-	86	I	88	100	IWMI	60	-	-
13	78	M	M	CP	-	-	-	92	I	80	96	ASMI	60	-	-
14	48	M	M	CP	-	-	-	100	I	100	160	ASMI	60	-	-
15	30	M	M	CP	-	-	-	80	I	68	120	IWMI	60	-	-
16	45	M	M	CP	-	-	-	77	I	74	129	AWMI	60	-	-
17	74	M	M	CP	-	-	-	60	I	88	112	ASMI	60	-	-
18	42	M	M	CP	-	-	-	70	I	80	120	IWMI	60	-	-
19	42	M	M	CP	-	-	-	84	I	66	86	ASMI	60	-	-
20	55	M	M	CP	-	-	-	66	I	80	100	AWMI	50	CCF	-

21	56	M	M	CP	-	-	-	80	I	78	99	IWMI	60	-	-
22	50	M	M	CP	-	-	-	-	IV			AWMI		CCF	+
23	47	M	M	CP	-	-	-	90	IV			ALMI		CCF	+
24	74	M	M	CP	-	-	-	60	I	106	165	IWMI	60	-	-
25	46	M	M	CP	-	-	-	92	I	80	100	IWMI	60	-	-
26	35	M	M	CP	-	-	-	80	I	108	128	ASMI	56	-	-
27	42	M	M	CP	-	-	-	80	I	66	78	ALMI	48	CCF	-
28	68	M	M	CP	-	-	-	180	I	60	74	ALMI	50	VT	-
29	53	M	M	CP	-	-	-	98	I	100	180	ASMI	57	-	-
30	79	M	M	SY	-	-	-	90	III	60	68	ALMI	50	CCF	-
31	40	M	M	CP	-	-	-	70	II	80	120	ASMI	54	CCF	-
32	67	M	M	CP	-	-	-	96	II	106	192	AWMI	50	CCF	-
33	59	F	M	BR	-	-	-	60	II	100	136	AWMI	35	CCF,CHB	+
34	42	M	M	CP	-	-	-	80	I	69	98	ASMI	60	-	-
35	60	M	M	CP	-	-	-	76	I	66	88	ASMI	60	-	-
36	43	M	M	CP	-	-	-	76	I	74	100	IWMI	57	-	-
37	45	M	S	CP	-	-	-	70	I	64	110	ASMI	60	-	-
38	47	M	M	CP	-	-	-	80	I	76	108	IWMI	56	-	-
39	40	M	M	CP	-	-	-	66	I	88	126	IWMI	60	-	-
40	44	M	S	CP	-	-	-	70	II	74	116	IWMI	47	CCF	+
41	60	M	M	CP	-	-	-	90	I	90	138	PWMI	56	-	-
42	44	M	M	CP	-	-	-	86	I	76	98	IWMI	60	-	-
43	43	M	M	CP	-	-	-	88	I	66	104	IWMI	60	-	-
44	52	M	M	CP	-	-	-	98	I	77	140	AWMI	54	-	-

45	70	M	M	CP	-	-	-	70	I	86	140	ASMI	56	-	-
46	57	M	M	CP	-	-	-	70	I	74	98	ASMI	56	-	-
47	50	M	M	CP	-	-	-	90	I	64	80	ASMI	58	-	-
48	60	M	M	CP	-	-	-	90	I	90	76	IWMI	60	-	-
49	50	M	M	CP	-	-	-	60	I	70	90	IWMI	60	-	-
50	55	M	M	CP	-	-	-	90	I	74	118	ASMI	59	-	-
51	56	M	M	BR	-	-	-	110	IV			AWMI		CCF	+
52	34	M	M	CP	-	-	-	86	I	66	78	ASMI	54	-	-
53	44	M	M	CP	-	-	-	88	I	68	78	IWMI	58	-	-
54	54	F	S	CP	-	-	-	70	I	98	130	IWMI	58	-	-
55	65	F	S	CP	-	-	-	80	I	90	130	IWMI	60	-	-
56	34	M	S	CP	-	-	-	80	II	54	77	ASMI	48	CCF	-
57	54	M	M	BR	-	-	-	110	IV			AWMI		CCF	+
58	42	M	S	CP	-	-	-	90	I	98	120	AWMI	50	-	-
59	37	F	S	CP	-	-	-	70	I	78	116	IWMI	58	-	-
60	60	M	M	CP	-	-	-	88	I	78	68	ASMI	58	-	-
61	42	M	S	CP	-	-	-	70	I	70	109	IWMI	60	-	-
62	67	M	M	BR	-	-	-	70	IV			AWMI		CCF	+
63	42	M	M	CP	-	-	-	90	I	68	79	ASMI	56	-	-
64	58	M	M	CP	-	-	-	90	I	90	130	IWMI	50	-	-
65	54	M	M	CP	-	-	-	80	I	84	118	IWMI	60	-	-
66	42	M	S	CP	-	-	-	90	I	78	106	IWMI	58	-	-
67	72	M	M	CP	-	-	-	90	I	88	120	IWMI	60	-	-
68	37	M	M	BR	-	-	-	100	IV			ASMI		CCF VF	+
69	75	M	S	CP	-	-	-	80	I	66	99	IWMI	60	-	-

70	50	M	M	CP	-	-	-	90	I	88	100	IWMI	58	-	-
71	42	M	M	CP	-	-	-	80	I	80	110	IWMI	60	-	-
72	55	M	M	CP	-	-	-	80	I	86	120	IWMI	60	-	-
73	75	F	S	CP	-	-	-	80	I	76	94	ASMI	56	-	-
74	34	M	S	CP	-	-	-	90	I	76	98	IWMI	60	-	-
75	57	M	M	CP	-	-	-	66	I	90	130	ASMI	58	-	-
76	67	M	M	CP	-	-	-	100	I	68	110	ASMI	56	-	-
77	65	M	M	CP	-	-	-	80	I	60	80	IWMI	60	-	-
78	38	M	S	CP	-	-	-	8O	I	86	120	ASMI	60	-	-
79	74	M	S	CP	-	-	-	90	I	100	168	ASMI	56	-	-
80	65	M	M	CP	-	-	-	80	I	77	106	AWMI	52	VT	-
81	45	M	M	CP	-	-	-	90	I	88	90	ASMI	60	-	-
82	67	F	M	CP	-	-	-	80	I	66	90	ASMI	58	-	-
83	43	M	M	CP	-	-	-	82	I	90	120	AWMI	54	-	-
84	38	M	S	CP	-	-	-	90	I	90	130	ASMI	60	-	-
85	50	M	M	CP	-	-	-	80	I	77	110	ASMI	60	-	-
86	53	M	S	CP	-	-	-	80	I	66	78	ASMI	60	-	-
87	70	M	M	CP	-	-	-	90	I	60	96	ASMI	58	-	-
88	52	M	M	CP	-	-	-	90	I	90	136	ASMI	58	-	-
89	36	M	M	CP	-	-	-	80	I	90	132	ASMI	60	-	-
90	50	F	S	CP	-	-	-	110	I	110	160	ALMI	60	-	-
91	43	M	M	C P	-	-	-	80	I	88	112	IWMI	60	-	-
92	66	M	S	CP	-	-	-	80	I	70	110	IWMI	60	-	-
93	70	M	M	CP	-	-	-	80	I	70	110	IWMI	60	-	-
94	53	M	M	CP	-	-	-	80	I	90	130	ASMI	56	-	-

95	47	M	M	CP	-	-	-	92	I	56	70	ASMI	60	-	-
96	48	M	S	CP	-	-	-	92	I	90	120	ASMI	60	-	-
97	37	F	S	CP	-	-	-	92	I	74	110	ASMI	58	-	-
98	35	M	M	CP	-	-	-	80	I	86	110	IWMI	60	-	-
99	43	M	M	CP	-	-	-	88	I	99	130	AWMI	54	-	-
100	68	M	M	CP	-	-	-	86	I	70	100	ASMI	60	-	-
101	70	M	M	CP	-	-	-	80	I	88	100	IWMI	60	-	-
102	65	F	M	CP	-	-	-	90	I	74	90	ASMI	60	-	-
103	65	F	S	CP	-	-	-	60	I	70	86	ASMI	57	-	-
104	50	M	M	CP	-	-	-	76	I	66	86	ALMI	60	-	-
105	40	M	M	CP	-	-	-	90	I	90	108	IWMI	60	-	-
106	45	M	M	CP	-	-	-	80	I	80	64	AWMI	59	-	-
107	62	M	M	CP	-	-	-	71	I	76	104	ASMI	60	-	-
108	54	M	S	CP	-	-	-	82	I	58	60	ASMI	59	-	-
109	37	M	S	CP	-	-	-	76	I	80	152	IWMI	60	-	-
110	52	M	M	CP	-	-	-	74	I	64	88	ASMI	60	-	-
111	54	M	M	CP	-	-	-	80	I	60	110	ASMI	58	-	-
112	45	M	S	CP	-	-	-	100	I	80	105	ALMI	60	-	-
113	50	M	M	CP	-	-	-	70	I	66	106	IWMI	60	-	-
114	47	M	M	CP	-	-	-	90	I	70	68	ALMI	60	-	-
115	83	M	S	CP	-	-	-	80	I	70	110	ALMI	60	-	-
116	53	M	M	CP	-	-	-	72	I	98	100	IWMI	57	-	-
117	40	M	M	CP	-	-	-	94	I	80	101	IWMI	60	-	-
118	45	M	M	CP	-	-	-	80	I	80	100	IWMI	60	-	-
119	80	F	S	CP	-	-	-	92	I	80	110	IWMI	60	-	-

120	65	F	M	CP	-	-	-	92	I	70	88	ASMI	56	-	-
121	69	F	S	CP	-	-	-	68	I	100	180	ALMI	57	-	-
122	78	F	S	CP	-	-	-	100	I	90	135	ASMI	57	-	-
123	60	F	S	CP	-	-	-	86	I	70	114	ASMI	60	-	-
124	63	M	M	CP	-	-	-	80	I	70	72	IWMI	60	-	-
125	40	F	S	CP	-	-	-	80	I	90	116	IWMI	60	-	-
126	50	M	M	CP	-	-	-	68	I	80	130	IWMI	60	-	-
127	58	M	S	CP	-	-	-	110	I	100	110	IWMI	60	-	-
128	34	M	M	CP	-	-	-	90	I	90	104	IWMI	60	-	-
129	55	M	M	CP	-	-	-	80	I	90	160	AWMI	60	-	-
130	60	M	M	CP	-	-	-	80	I	60	75	ASMI	60	-	-
131	27	M	S	CP	-	-	-	80	I	66	100	IWMI	60	-	-
132	60	M	M	CP	-	-	-	80	I	60	78	ASMI	58	-	-
133	57	M	M	CP	-	-	-	62	I	78	98	ASMI	60	-	-
134	68	M	M	CP	-	-	-	80	I	90	127	ASMI	60	-	-
135	49	M	M	CP	-	-	-	88	I	70	50	ASMI	60	-	-
136	65	M	M	CP	-	-	-	90	I	90	170	ASMI	60	-	-
137	70	M	M	CP	-	-	-	89	I	70	88	ASMI	60	-	-
138	45	M	M	CP	-	-	-	87	I	68	110	IWMI	60	-	-
139	39	M	M	CP	-	-	-	92	I	80	78	IWMI	60	-	-
140	56	M	M	CP	-	-	-	84	II	80	110	ASMI	56	CCF	-
141	52	M	M	CP	-	-	-	54	I	90	130	IWMI	58	-	-
142	45	F	S	CP	-	-	-	80	I	66	119	AWMI	60	-	-
143	74	M	M	CP	-	-	-	85	I	68	75	IWMI	58	-	-
144	70	M	M	CP	-	-	-	76	I	78	120	ASMI	56	-	-

145	50	M	M	CP	-	-	-	66	I	76	94	ALMI	58	-	-
146	68	M	M	CP	-	-	-	92	I	100	176	ASMI	58	-	-
147	44	M	M	CP	-	-	-	86	I	76	120	ASMI	60	-	-
148	38	M	M	CP	-	-	-	98	I	97	164	IWMI	60	-	-
149	52	M	M	CP	-	-	-	118	I	98	120	IWMI	60	-	-
150	37	F	S	CP	-	-	-	86	I	70	98	ALMI	60	-	-
151	52	M	M	CP	-	-	-	86	I	66	83	ASMI	58	-	-
152	76	M	M	CP	-	-	-	80	I	88	110	IWMI	54	-	-
153	38	M	S	CP	-	-	-	94	I	98	124	IWMI	60	-	-
154	45	M	M	CP	-	-	-	86	I	77	102	AWMI	58	-	-
155	70	M	M	CP	-	-	-	87	I	90	180	AWMI	56	-	-
156	53	M	M	CP	-	-	-	60	I	96	150	IWMI	56	-	-
157	51	M	M	CP	-	-	-	64	I	98	188	ASMI	60	-	-
158	55	F	S	CP	-	-	-	82	I	75	128	IWMI	60	-	-
159	55	F	S	CP	-	-	-	86	I	52	71	IWMI	58	-	-
160	62	F	S	CP	-	-	-	106	II	64	70	AWMI	56	CCF	-
161	51	F	M	CP	-	-	-	82	I	96	192	ASMI	60	-	-
162	74	M	S	CP	-	-	-	88	I	96	110	ASMI	58	-	-
163	41	M	M	CP	-	-	-	102	I	60	58	IWMI	60	-	-
164	59	M	M	CP	-	-	-	64	I	54	75	ASMI	56	-	-
165	53	M	M	CP	-	-	-	92	I	66	60	IWMI	60	-	-
166	56	M	M	CP	-	-	-	92	I	68	54	IWMI	60	-	-
167	72	M	M	CP	-	-	-	70	I	66	91	ASMI	60	-	-
168	56	M	M	CP	-	-	-	66	I	76	90	ASMI	60	-	-
169	40	M	M	CP	-	-	-	100	I	56	60	IWMI	60	-	-

170	49	M	M	CP	-	-	-	104	II	64	90	AWMI	50	CCF	-
171	40	M	M	CP	-	-	-	100	I	68	99	IWMI	60	-	-
172	39	M	S	CP	-	-	-	62	I	65	125	IWMI	60	-	-
173	41	M	M	CP	-	-	-	70	I	96	128	IWMI	60	-	-
174	48	M	M	CP	-	-	-	84	I	68	76	IWMI	60	-	-
175	50	M	M	CP	-	-	-	57	I	76	120	IWMI	60	-	-
176	35	M	S	CP	-	-	-	82	I	56	65	IWMI	58	-	-
177	28	M	S	CP	-	-	-	92	I	96	125	ASMI	60	-	-
178	56	M	M	CP	-	-	-	120	I	90	176	ASMI	60	-	-
179	55	F	M	CP	-	-	-	78	I	99	140	ALMI	60	-	-
180	60	F	S	CP	-	-	-	88	I	76	88	IWMI	60	-	-
181	38	F	S	CP	-	-	-	76	I	86	89	IWMI	60	-	-
182	60	M	M	CP	-	-	-	76	I	80	100	IWMI	60	-	-
183	60	M	S	SY	-	-	-	72	I	110	194	ASMI	60	-	-
184	58	M	M	CP	-	-	-	56	I	87	116	IWMI	57	-	-
185	74	M	M	CP	-	-	-	66	I	56	78	IWMI	58	-	-
186	65	F	S	CP	-	-	-	88	I	98	104	IWMI	60	-	-
187	46	M	S	CP	-	-	-	68	I	67	93	ASMI	58	-	-
188	54	M	M	CP	-	-	-	76	I	90	124	ASMI	60	-	-
189	67	M	M	CP	-	-	-	80	I	98	110	ASMI	56	-	-
190	54	F	S	CP	-	-	-	64	I	67	88	ASMI	60	-	-
191	56	M	M	CP	-	-	-	92	I	68	54	IWMI	60	-	-
192	72	M	M	CP	-	-	-	70	I	66	91	ASMI	60	-	-
193	56	M	M	CP	-	-	-	66	I	76	90	ASMI	60	-	-
194	40	M	M	CP	-	-	-	100	I	56	60	IWMI	60	-	-

195	49	M	M	CP	-	-	-	104	II	64	90	AWMI	50	CCF	-
196	40	M	M	CP	-	-	-	100	I	68	99	IWMI	60	-	-
197	39	M	S	CP	-	-	-	62	I	65	125	IWMI	60	-	-
198	41	M	M	CP	-	-	-	70	I	96	128	IWMI	60	-	-
199	48	M	M	CP	-	-	-	84	I	68	76	IWMI	60	-	-
200	50	M	M	CP	-	-	-	57	I	76	120	IWMI	60	-	-
201	35	M	S	CP	-	-	-	82	I	56	65	IWMI	58	-	-
202	28	M	S	CP	-	-	-	92	I	96	125	ASMI	60	-	-
203	56	M	M	CP	-	-	-	120	I	90	176	ASMI	60	-	-
204	55	F	M	CP	-	-	-	78	I	99	140	ALMI	60	-	-
205	60	F	S	CP	-	-	-	88	I	76	88	IWMI	60	-	-
206	38	F	S	CP	-	-	-	76	I	86	89	IWMI	60	-	-
207	60	M	M	CP	-	-	-	76	I	80	100	IWMI	60	-	-
208	60	M	S	SY	-	-	-	72	I	110	194	ASMI	60	-	-
209	58	M	M	CP	-	-	-	56	I	87	116	IWMI	57	-	-
210	74	M	M	CP	-	-	-	66	I	56	78	IWMI	58	-	-
211	65	F	S	CP	-	-	-	88	I	98	104	IWMI	60	-	-
212	46	M	S	CP	-	-	-	68	I	67	93	ASMI	58	-	-
213	54	M	M	CP	-	-	-	76	I	90	124	ASMI	60	-	-
214	67	M	M	CP	-	-	-	80	I	98	110	ASMI	56	-	-
215	54	F	S	CP	-	-	-	64	I	67	83	IWMI	60	-	-
216	53	M	M	CP	-	-	-	92	I	66	60	IWMI	60	-	-
217	42	M	M	CP	-	-	-	80	I	66	78	ALMI	48	CCF	-
218	70	M	M	CP	-	-	-	87	I	90	180	AWMI	56	-	-
219	68	M	M	CP	-	-	-	180	I			ALMI		VF	+

220	53	M	M	CP	-	-	-	98	I	100	180	ASMI	57	-	-
221	79	M	M	SY	-	-	-	90	III	60	68	ALMI	50	CCF	-
222	40	M	M	CP	-	-	-	70	II	80	120	ASMI	54	CCF	-
223	67	M	M	CP	-	-	-	96	II	106	192	AWMI	50	CCF	-
224	59	F	M	BR	-	-	-	60	II	100	136	AWMI	35	CCF CHB	+
225	42	M	M	CP	-	-	-	80	I	69	98	ASMI	60	-	-
226	60	M	M	CP	-	-	-	76	I	66	88	ASMI	60	-	-
227	43	M	M	CP	-	-	-	76	I	74	100	IWMI	57	-	-
228	45	M	S	CP	-	-	-	70	I	64	110	ASMI	60	-	-
229	47	M	M	CP	-	-	-	80	I	76	108	IWMI	56	-	-
230	40	M	M	CP	-	-	-	66	I	88	126	IWMI	60	-	-
231	44	M	S	CP	-	-	-	70	II	74	116	IWMI	47	CCF	+
232	60	M	M	CP	-	-	-	90	I	90	138	PWMI	56	-	-
233	44	M	M	CP	-	-	-	86	I	76	98	IWMI	60	-	-
234	43	M	M	CP	-	-	-	88	I	66	104	IWMI	60	-	-
235	52	M	M	CP	-	-	-	98	I	77	140	AWMI	54	-	-
236	70	M	M	CP	-	-	-	70	I	86	140	ASMI	56	-	-
237	57	M	M	CP	-	-	-	70	I	74	98	ASMI	56	-	-
238	50	M	M	CP	-	-	-	90	I	64	80	ASMI	58	-	-
239	60	M	M	CP	-	-	-	90	I	90	76	IWMI	60	-	-
240	50	M	M	CP	-	-	-	60	I	70	90	IWMI	60	-	-
241	55	M	M	CP	-	-	-	90	I	74	118	ASMI	59	-	-
242	56	M	M	BR	-	-	-	110	IV			AWMI		CCF VF	+
243	34	M	M	CP	-	-	-	86	I	66	78	ASMI	54	-	-

244	44	M	M	CP	-	-	-	88	I	68	78	IWMI	58	-	-
245	54	F	S	CP	-	-	-	70	I	98	130	IWMI	58	-	-
246	65	F	S	CP	-	-	-	80	I	90	130	IWMI	60	-	-
247	34	M	S	CP	-	-	-	80	II	54	77	ASMI	48	CCF	-
248	54	M	M	BR	-	-	-	110	IV			AWMI		CCF	+
249	42	M	S	CP	-	-	-	90	I	98	120	AWMI	50	-	-
250	37	F	S	CP	-	-	-	70	I	78	116	IWMI	58	-	-
251	60	M	M	CP	-	-	-	88	I	78	68	ASMI	58	-	-
252	42	M	S	CP	-	-	-	70	I	70	109	IWMI	60	-	-
253	67	M	M	BR	-	-	-	70	IV			AWMI		CCF	+
254	42	M	M	CP	-	-	-	90	I	68	79	ASMI	56	-	-
255	58	M	M	CP	-	-	-	90	I	90	130	IWMI	50	-	-
256	54	M	M	CP	-	-	-	80	I	84	118	IWMI	60	-	-
257	42	M	S	CP	-	-	-	90	I	78	106	IWMI	58	-	-
258	72	M	M	CP	-	-	-	90	I	88	120	IWMI	60	-	-
259	37	M	M	BR	-	-	-	100	IV			ASMI		CCF	+
260	75	M	S	CP	-	-	-	80	I	66	99	IWMI	60	-	-
261	50	M	M	CP	-	-	-	90	I	88	100	IWMI	58	-	-
262	42	M	M	CP	-	-	-	80	I	80	110	IWMI	60	-	-
263	55	M	M	CP	-	-	-	80	I	86	120	IWMI	60	-	-
264	75	F	S	CP	-	-	-	80	I	76	94	ASMI	56	-	-
265	34	M	S	CP	-	-	-	90	I	76	98	IWMI	60	-	-
266	57	M	M	CP	-	-	-	66	I	90	130	ASMI	58	-	-
267	67	M	M	CP	-	-	-	100	I	68	110	ASMI	56	-	-
268	65	M	M	CP	-	-	-	80	I	60	80	IWMI	60	-	-

269	38	M	S	CP	-	-	-	8O	I	86	120	ASMI	60	-	-
270	74	M	S	CP	-	-	-	90	I	100	168	ASMI	56	-	-
271	65	M	M	CP	-	-	-	80	I	77	106	AWMI	52	VT	-
272	45	M	M	CP	-	-	-	90	I	88	90	ASMI	60	-	-
273	67	F	M	CP	-	-	-	80	I	66	90	ASMI	58	-	-
274	43	M	M	CP	-	-	-	82	I	90	120	AWMI	54	-	-
275	38	M	S	CP	-	-	-	90	I	90	130	ASMI	60	-	-
276	50	M	M	CP	-	-	-	80	I	77	110	ASMI	60	-	-
277	53	M	S	CP	-	-	-	80	I	66	78	ASMI	60	-	-
278	70	M	M	CP	-	-	-	90	I	60	96	ASMI	58	-	-
279	52	M	M	CP	-	-	-	90	I	90	136	ASMI	58	-	-
280	36	M	M	CP	-	-	-	80	I	90	132	ASMI	60	-	-
281	50	F	S	CP	-	-	-	110	I	110	160	ALMI	60	-	-
282	43	M	M	C P	-	-	-	80	I	88	112	IWMI	60	-	-
283	66	M	S	CP	-	-	-	80	I	70	110	IWMI	60	-	-
284	70	M	M	CP	-	-	-	80	I	70	110	IWMI	60	-	-
285	53	M	M	CP	-	-	-	80	I	90	130	ASMI	56	-	-
286	47	M	M	CP	-	-	-	92	I	56	70	ASMI	60	-	-
287	48	M	S	CP	-	-	-	92	I	90	120	ASMI	60	-	-
288	37	F	S	CP	-	-	-	92	I	74	110	ASMI	58	-	-
289	35	M	M	CP	-	-	-	80	I	86	110	IWMI	60	-	-
290	43	M	M	CP	-	-	-	88	I	99	130	AWMI	54	-	-
291	68	M	M	C P	-	-	-	86	I	70	100	ASMI	60	-	-
292	70	M	M	CP	-	-	-	80	I	88	100	IWMI	60	-	-
293	65	F	M	CP	-	-	-	90	I	74	90	ASMI	60	-	-

294	65	F	S	CP	-	-	-	60	I	70	86	ASMI	57	-	-
295	50	M	M	CP	-	-	-	76	I	66	86	ALMI	60	-	-
296	40	M	M	CP	-	-	-	90	I	90	108	IWMI	60	-	-
297	45	M	M	CP	-	-	-	80	I	80	64	AWMI	59	-	-
298	62	M	M	CP	-	-	-	71	I	76	104	ASMI	60	-	-
299	54	M	S	CP	-	-	-	82	I	58	60	ASMI	59	-	-
300	37	M	S	CP	-	-	-	76	I	80	152	IWMI	60	-	-
301	52	M	M	CP	-	-	-	74	I	64	88	ASMI	60	-	-
302	54	M	M	CP	-	-	-	80	I	60	110	ASMI	58	-	-
303	45	M	S	CP	-	-	-	100	I	80	105	ALMI	60	-	-
304	50	M	M	CP	-	-	-	70	I	66	106	IWMI	60	-	-
305	47	M	M	CP	-	-	-	90	I	70	68	ALMI	60	-	-
306	83	M	S	CP	-	-	-	80	I	70	110	ALMI	60	-	-
307	53	M	M	CP	-	-	-	72	I	98	100	IWMI	57	-	-
308	40	M	M	CP	-	-	-	94	I	80	101	IWMI	60	-	-
309	45	M	M	CP	-	-	-	80	I	80	100	IWMI	60	-	-
310	80	F	S	CP	-	-	-	92	I	80	110	IWMI	60	-	-
311	65	F	M	CP	-	-	-	92	I	70	88	ASMI	56	-	-
312	69	F	S	CP	-	-	-	68	I	100	180	ALMI	57	-	-
313	78	F	S	CP	-	-	-	100	I	90	135	ASMI	57	-	-
314	60	F	S	CP	-	-	-	86	I	70	114	ASMI	60	-	-
315	63	M	M	CP	-	-	-	80	I	70	72	IWMI	60	-	-
316	40	F	S	CP	-	-	-	80	I	90	116	IWMI	60	-	-
317	50	M	M	CP	-	-	-	68	I	80	130	IWMI	60	-	-
318	58	M	S	CP	-	-	-	110	I	100	110	IWMI	60	-	-

319	34	M	M	CP	-	-	-	90	I	90	104	IWMI	60	-	-
320	55	M	M	CP	-	-	-	80	I	90	160	AWMI	60	-	-
321	60	M	M	CP	-	-	-	80	I	60	75	ASMI	60	-	-
322	27	M	S	CP	-	-	-	80	I	66	100	IWMI	60	-	-
323	60	M	M	CP	-	-	-	80	I	60	78	ASMI	58	-	-
324	57	M	M	CP	-	-	-	62	I	78	98	ASMI	60	-	-
325	68	M	M	CP	-	-	-	80	I	90	127	ASMI	60	-	-
326	49	M	M	CP	-	-	-	88	I	70	50	ASMI	60	-	-
327	65	M	M	CP	-	-	-	90	I	90	170	ASMI	60	-	-
328	70	M	M	CP	-	-	-	89	I	70	88	ASMI	60	-	-
329	45	M	M	CP	-	-	-	87	I	68	110	IWMI	60	-	-
330	39	M	M	CP	-	-	-	92	I	80	78	IWMI	60	-	-
331	56	M	M	CP	-	-	-	84	II	80	110	ASMI	56	CCF	-
332	52	M	M	CP	-	-	-	54	I	90	130	IWMI	58	-	-
333	45	F	S	CP	-	-	-	80	I	66	119	AWMI	60	-	-
334	74	M	M	CP	-	-	-	85	I	68	75	IWMI	58	-	-
335	70	M	M	CP	-	-	-	76	I	78	120	ASMI	56	-	-
336	50	M	M	CP	-	-	-	66	I	76	94	ALMI	58	-	-
337	68	M	M	CP	-	-	-	92	I	100	176	ASMI	58	-	-
338	44	M	M	CP	-	-	-	86	I	76	120	ASMI	60	-	-
339	38	M	M	CP	-	-	-	98	I	97	164	IWMI	60	-	-
340	52	M	M	CP	-	-	-	118	I	98	120	IWMI	60	-	-
341	37	F	S	CP	-	-	-	86	I	70	98	ALMI	60	-	-
342	52	M	M	CP	-	-	-	86	I	66	83	ASMI	58	-	-
343	76	M	M	CP	-	-	-	80	I	88	110	IWMI	54	-	-

344	38	M	S	CP	-	-	-	94	I	98	124	IWMI	60	-	-
345	45	M	M	CP	-	-	-	86	I	77	102	AWMI	58	-	-
346	53	M	M	CP	-	-	-	60	I	96	150	IWMI	56	-	-
347	51	M	M	CP	-	-	-	64	I	98	188	ASMI	60	-	-
348	55	F	S	CP	-	-	-	82	I	75	128	IWMI	60	-	-
349	55	F	S	CP	-	-	-	86	I	52	71	IWMI	58	-	-
350	62	F	S	CP	-	-	-	106	II	64	70	AWMI	56	CCF	-
351	51	F	M	CP	-	-	-	82	I	96	192	ASMI	60	-	-
352	74	M	S	CP	-	-	-	88	I	96	110	ASMI	58	-	-
353	41	M	M	CP	-	-	-	102	I	60	58	IWMI	60	-	-
354	59	M	M	CP	-	-	-	64	I	54	75	ASMI	56	-	-
355	65	F	S	CP	-	-	-	82	II	78	120	ASMI	60	CCF	-
356	62	F	S	CP	-	-	-	60	I	90	110	IWMI	60	-	-
357	61	F	S	CP	-	-	-	88	I	80	90	ALMI	6O	-	-
358	35	M	M	CP	-	-	-	80	I	74	86	ASMI	54	-	-
359	46	M	M	CP	-	-	-	76	I	80	120	ASMI	60	-	-
360	44	M	S	CP	-	-	-	80	I	80	100	ASMI	60	-	-
361	60	M	M	CP	-	-	-	8O	I	64	124	AWMI	50	-	-
362	48	M	M	CP	-	-	-	90	I	80	125	ASMI	60	-	-
363	70	M	S	CP	-	-	-	84	I	110	136	ASMI	60	-	-
364	48	M	M	CP	-	-	-	90	I	70	100	ALMI	60	-	-
365	59	F	M	CP	-	-	-	80	I	74	128	IWMI	62	-	-
366	54	M	M	CP	-	-	-	86	I	88	100	IWMI	60	-	-
367	78	M	M	CP	-	-	-	92	I	80	96	ASMI	60	-	-
368	48	M	M	CP	-	-	-	100	I	100	160	ASMI	60	-	-

369	30	M	M	CP	-	-	-	80	I	68	120	IWMI	60	-	-
370	45	M	M	CP	-	-	-	77	I	74	129	AWMI	60	-	-
371	74	M	M	CP	-	-	-	60	I	88	112	ASMI	60	-	-
372	42	M	M	CP	-	-	-	70	I	80	120	IWMI	60	-	-
373	42	M	M	CP	-	-	-	84	I	66	86	ASMI	60	-	-
374	55	M	M	CP	-	-	-	66	I	80	100	AWMI	50	CCF	-
375	56	M	M	CP	-	-	-	80	I	78	99	IWMI	60	-	-
376	50	M	M	CP	-	-	-	64	IV			AWMI		CCF	+
377	47	M	M	CP	-	-	-	90	IV			ALMI		CCF	+
378	74	M	M	CP	-	-	-	60	I	106	165	IWMI	60	-	-
379	46	M	M	CP	-	-	-	92	I	80	100	IWMI	60	-	-
380	35	M	M	CP	-	-	-	80	I	108	128	ASMI	56	-	-
381	65	F	S	CP	-	-	-	82	II	78	120	ASMI	60	CCF	-
382	62	F	S	CP	-	-	-	60	I	90	110	IWMI	60	-	-
383	61	F	S	CP	-	-	-	88	I	80	90	ALMI	6O	-	-
384	35	M	M	CP	-	-	-	80	I	74	86	ASMI	54	-	-
385	46	M	M	CP	-	-	-	76	I	80	120	ASMI	60	-	-
386	44	M	S	CP	-	-	-	80	I	80	100	ASMI	60	-	-
387	60	M	M	CP	-	-	-	8O	I	64	124	AWMI	50	-	-
388	48	M	M	CP	-	-	-	90	I	80	125	ASMI	60	-	-
389	70	M	S	CP	-	-	-	84	I	110	136	ASMI	60	-	-
390	48	M	M	CP	-	-	-	90	I	70	100	ALMI	60	-	-
391	59	F	M	CP	-	-	-	80	I	74	128	IWMI	62	-	-
392	54	M	M	CP	-	-	-	86	I	88	100	IWMI	60	-	-
393	78	M	M	CP	-	-	-	92	I	80	96	ASMI	60	-	-

394	48	M	M	CP	-	-	-	100	I	100	160	ASMI	60	-	-
395	30	M	M	CP	-	-	-	80	I	68	120	IWMI	60	-	-
396	45	M	M	CP	-	-	-	77	I	74	129	AWMI	60	-	-
397	74	M	M	CP	-	-	-	60	I	88	112	ASMI	60	-	-
398	42	M	M	CP	-	-	-	70	I	80	120	IWMI	60	-	-
399	42	M	M	CP	-	-	-	84	I	66	86	ASMI	60	-	-
400	55	M	M	CP	-	-	-	66	I	80	100	AWMI	50	CCF	-
401	56	M	M	CP	-	-	-	80	I	78	99	IWMI	60	-	+
402	50	M	M	CP	-	-	-	76	IV			AWMI		CCF	+
403	47	M	M	CP	-	-	-	90	IV			ALMI		CCF	+
404	74	M	M	CP	-	-	-	60	I	106	165	IWMI	60	-	-
405	46	M	M	CP	-	-	-	92	I	80	100	IWMI	60	-	-
406	35	M	M	CP	-	-	-	80	I	108	128	ASMI	56	-	-
407	46	M	S	CP	+	3	IR	98	IV			AWMI		CCF	+
408	58	F	S	CP	+	7	IR	92	I			IWMI		VF	+
409	65	F	M	CP	+	8	R	80	I	186	320	ASMI	56	-	-
410	58	F	M	CP	+	8	R	90	II	130	188	ASMI	54	CCF	-
411	60	F	S	CP	+	4	R	86	I	168	200	ASMI	60	-	-
412	51	M	M	CP	+	3	IR	52	I	200	390	ASMI	60	-	-
413	35	F	S	CP	+	3	IR	80	I	219	300	ASMI	56	-	-
414	65	M	M	CP	+	4	R	88	I	130	170	ASMI	60	-	-
415	49	F	S	CP	+	1	R	90	I	88	168	ALMI	60	-	-
416	48	M	M	CP	+	7	IR	80	I	260	384	ASMI	60	-	-
417	55	M	M	BR	+	9	R	100	III	96	118	AWMI	45	CCF	-
418	65	M	M	BR	+	4	R	110	III			AWMI	30	CCF,VF	+

419	37	M	S	CP	+	3	IR	80	I	168	204	ASMI	50	-	-
420	52	M	M	BR	+	8	R	90	II	140	210	AWMI	44	CCF	-
421	40	M	S	BR	+	4	IR	96	II	198	278	AWMI	36	CCF	-
422	34	M	S	CP	+	3	R	90	I	288	348	ASMI	50	CHB	+
423	45	M	S	CP	+	4	IR	80	II	190	270	AWMI	45	CCF	-
424	59	M	M	CP	+	9	R	70	I	170	230	IWMI	54	-	-
425	63	M	M	BR	+	8	IR	110	II	210	328	AWMI	40	CCF CHB	+
426	53	F	S	CP	+	2	R	90	I	66	78	AWMI	50	-	-
427	67	F	S	BR	+	3	IR	99	II	186	270	AWMI	44	CCF	-
428	44	M	M	CP	+	8	R	80	I	99	140	ASMI	56	-	-
429	68	M	M	CP	+	7	IR	90	II	216	320	AWMI	44	CCF	-
430	68	F	S	BR	+	3	IR	90	II	230	290	AWMI	35	CCF	-
431	49	M	M	CP	+	3	R	86	I	136	210	ASMI	50	-	-
432	66	F	S	CP	+	4	R	90	I	160	236	IWMI	56	-	-
433	55	F	S	CP	+	8	R	102	II	240	388	ASMI	48	CCF	-
434	65	M	M	CP	+	3	R	80	I	168	220	ASMI	52	-	-
435	50	M	S	CP	+	6	R	86	II	210	260	ALMI	50	CCF	-
436	34	M	M	CP	+	1	R	72	I	140	240	ASMI	54	-	-
437	54	M	S	CP	+	3	R	70	I	130	160	AWMI	50	-	-
438	40	M	S	CP	+	4	R	92	II	180	234	AWMI	40	CCF	-
439	65	F	S	BR	+	9	IR	66	II	126	190	ASMI	48	-	-
440	54	M	S	CP	+	6	IR	86	I	177	280	ASMI	50	CCF	-
441	47	M	M	CP	+	4	IR	80	I	280	354	ASMI	56	-	-
442	40	M	M	CP	+	7	IR	88	I	130	270	IWMI	56	-	-

443	40	M	M	CP	+	8	IR	90	II	190	270	AWMI	42	CCF	-
444	54	M	M	CP	+	1	IR	94	I	160	171	AWMI	46	CCF	-
445	61	F	S	BR	+	4	IR	80	II	230	216	AWMI	35	CCF	-
446	57	M	M	CP	+	8	R	89	I	93	196	ASMI	50	-	-
447	72	F	S	CP	+	3	IR	98	II	260	290	ALMI	54	-	-
448	79	M	M	CP	+	8	R	100	I	130	218	ALMI	54	-	-
449	63	M	S	BR	+	7	R	88	IV			ALMI		CCF VF	+
450	75	F	S	CP	+	4	IR	60	I	130	210	AWMI	50	CHB	-
451	70	F	S	CP	+	8	IR	88	I	170	201	AWMI	50	CHB	-
452	55	M	M	CP	+	2	R	50	I	190	230	IWMI	58	-	-
453	53	M	M	CP	+	2	R	84	I	130	160	AWMI	50	-	-
454	51	M	M	CP	+	3	R	78	I	180	232	ASMI	50	-	-
455	64	M	M	CP	+	9	IR	80	I	200	195	ASMI	56	-	-
456	72	M	M	CP	+	1	IR	88	I	200	280	ASMI	56	-	-
457	63	M	M	CP	+	7	IR	82	I	280	394	IWMI	56	-	-
458	39	M	M	BR	+	4	R	78	II	180	240	IWMI	50	CCF CHB	+
459	74	M	M	CP	+	4	R	84	II	118	136	ASMI	50	CCF	-
460	62	M	M	CP	+	3	IR	86	I	310	358	ASMI	56	-	-
461	52	M	M	BR	+	3	IR	78	I	266	320	IWMI	56	-	-
462	58	F	M	CP	+	4	R	82	I	128	240	IWMI	58	-	-
463	47	M	M	CP	+	1	IR	86	I	189	225	AWMI	50	-	-
464	55	F	S	CP	+	13	R	88	II	200	288	AWMI	48	CCF	-
465	56	M	M	CP	+	9	R	86	II	140	226	ASMI	57	CCF	-
466	44	M	M	CP	+	3	IR	100	II	200	310	AWMI	40	CCF	-

467	70	M	M	CP	+	3	IR	78	I	150	210	AWMI	54	-	-
468	70	M	S	CP	+	4	R	90	I	170	326	AWMI	54	-	-
469	41	F	S	CP	+	3	IR	99	I	126	400	ASMI	54	-	-
470	71	F	S	BR	+	12	IR	74	II	210	316	AWMI	48	CCF VT	-
471	47	M	M	CP	+	7	R	110	II	190	270	AWMI	50	CCF	-
472	51	M	M	CP	+	3	IR	52	I	200	390	ASMI	60	-	-
473	35	F	S	CP	+	3	IR	80	I	219	300	ASMI	56	-	-
474	65	M	M	CP	+	4	R	88	I	130	170	ASMI	60	-	-
475	49	F	S	CP	+	1	R	90	I	88	168	ALMI	60	-	-
476	48	M	M	CP	+	7	IR	80	I	260	384	ASMI	60	-	-
477	55	M	M	BR	+	9	R	100	III	96	118	AWMI	45	CCF	-
478	65	M	M	BR	+	4	R	110	IV			AWMI		CCF,VF	+
479	37	M	S	CP	+	3	IR	80	I	168	204	ASMI	50	-	-
480	52	M	M	BR	+	8	R	90	II	140	210	AWMI	44	CCF	-
481	40	M	S	BR	+	4	IR	96	IV			AWMI		CCF	+
482	34	M	S	CP	+	3	R	90	I	288	348	ASMI	50	CHB	+
483	45	M	S	CP	+	4	IR	80	II	190	270	AWMI	45	CCF	-
484	59	M	M	CP	+	9	R	70	I	170	230	IWMI	54	-	-
485	63	M	M	BR	+	8	IR	110	II	210	328	AWMI	40	CCF CHB	+
486	53	F	S	CP	+	2	R	90	I	66	78	AWMI	50	-	-
487	67	F	S	BR	+	3	IR	99	II	186	270	AWMI	44	CCF	-
488	44	M	M	CP	+	8	R	80	I	99	140	ASMI	56	-	-
489	68	M	M	CP	+	7	IR	90	II	216	320	AWMI	44	CCF	-
490	68	F	S	BR	+	3	IR	90	IV			AWMI		CCF	+

491	49	M	M	CP	+	3	R	86	I	136	210	ASMI	50	-	-
492	66	F	S	CP	+	4	R	90	I	160	236	IWMI	56	-	-
493	55	F	S	CP	+	8	R	102	II	240	388	ASMI	48	CCF	-
494	65	M	M	CP	+	3	R	80	I	168	220	ASMI	52	-	-
495	50	M	S	CP	+	6	R	86	II	210	260	ALMI	50	CCF	-
496	34	M	M	CP	+	1	R	72	I	140	240	ASMI	54	-	-
497	54	M	S	CP	+	3	R	70	I	130	160	AWMI	50	-	-
498	40	M	S	CP	+	4	R	92	II	180	234	AWMI	40	CCF	-
499	65	F	S	BR	+	9	IR	66	II	126	190	ASMI	48	-	-
500	54	M	S	CP	+	6	IR	86	I	177	280	ASMI	50	CCF	-
501	46	M	S	CP	+	3	IR	98	IV			AWMI		CCF	+
502	58	F	S	CP	+	7	IR	92	I			IWMI		VF	+
503	65	F	M	CP	+	8	R	80	I	186	320	ASMI	56	-	-
504	58	F	M	CP	+	8	R	90	II	130	188	ASMI	54	CCF	-
505	63	M	M	CP	+	4	IR	82	I	280	394	IWMI	56	-	-
506	39	M	M	CP	+	3	R	80	II	180	240	IWMI	50	CHB	-
507	74	M	M	CP	+	4	R	84	II	118	136	ASMI	50	-	-
508	52	M	M	BR	+	3	IR	86	I	266	320	IWMI	56	-	-
509	58	F	M	CP	+	4	R	82	I	128	240	IWMI	58	-	-
510	54	M	M	CP	+	4	IR	86	I	189	225	AWMI	50	-	-
511	47	M	M	CP	+	7	R	110	IV			AWMI		CCF	+
512	56	M	M	CP	+	3	IR	98	I	136	200	IWMI	54	-	-
513	71	F	S	BR	+	9	IR	74	IV			AWMI		CCF VT	+
514	41	M	M	CP	+	2	IR	99	I	126	400	ASMI	54	-	-
515	58	M	S	CP	+	1	R	90	I	170	326	AWMI	54	-	-

516	70	M	M	CP	+	1	IR	78	I	150	210	AWMI	54	-	-
517	44	M	M	CP	+	2	IR	100	II			AWMI		CCF	+
518	56	M	M	CP	+	8	R	86	II	140	226	ASMI	57	-	-
519	62	M	M	CP	+	3	IR	88	I	310	358	ASMI	56	-	-